# FEAR : MEASUREMENT AND MODIFICATION

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## ABSTRACT

The avoidance and response prevention (RP) literatures are reviewed. This review highlighted a number of persistent issues, revealed a need for the development of sensitive fear assessment measures and showed a number of parameters that enhance response prevention's efficacy in reducing conditioned fear. This thesis examined (i) a number of RP parameters across escape-from-fear and passive avoidance baselines, using in the latter situation, multivariate fear assessment measures; and (ii) the psychopharmacological actions of the benzodiazepine, diazepam, and beta-adrenergic blockers, propranolol and atenolol, across passive avoidance, conditioned suppression of licking and signal detection behavioural baselines.

In experiment one, escape-from-fear behaviour was found to be insensitive to 100 - and 200 - non-reinforced 5 second CS presentations (RP). Massed RP was reported to be superior to distributed RP and protracted RP (2 hr.) more efficacious than 1 hr. RP in reducing conditioned fear and hastening avoidance extinction in experiment two. Social facilitation of RP (experiment three) enhanced RP effects when assessed by the time fear assessment measure but had less effect on RP when assessed by the approaches and first entry latency measures. This differential sensitivity of the fear assessment measures was also reported for diazepam, propranolol and atenolol - assisted RP (experiments three and four).

Experiment five examined the separate and combined administration of diazepam and propranolol on disinhibiting

licking behaviour. Diazepam was more effective than the combined treatment, which in turn was more effective than propranolol alone, with atenolol having little effect in disinhibiting licking behaviour.

Experiment six established a signal detection behavioural baseline which was used to independently assess possible diazepam - and propranolol-induced changes in stimulus discrimination from possible drug-induced changes in response-bias (experiment seven). Both diazepam and propranolol failed to affect response bias, whereas stimulus discrimination was attenuated by propranolol but unaffected by diazepam administration.

The response prevention results were discussed in terms of a modified two process theory presented in chapter two, which emphasised the relative sensitivity of and the relationship between the fear assessment measures in discriminating RP effects on conditioned fear and avoidance behaviour. The drug results were discussed in terms of the respective modes of anxiolytic action of the benzodiazepines and beta-adrenergic blockers. The signal detection results were discussed in terms of matching model and signal detection analyses of choice behaviour. Implications of these results to avoidance theory and research as well as to the assessment and treatment of fear motivated human neurotic behaviours were discussed with suggestions for future research being presented.

## OVERVIEW

Behaviour change in the laboratory as well as the real world can take place through the use of one of two distinct conditioning procedures, classical (respondent) or instrumental (operant) conditioning, or the combination of the two, as in avoidance learning, or through mediated learning processes (Kalish, 1981). Skinner (1938) distinguished between classical and instrumental conditioning as follows:

> The essence of (classical conditioning) is the substitution of one stimulus (the CS) for another (the US), or, as Pavlov put it, signalization. It prepares the organism by obtaining the elicitation of a response before the original stimulus (the US, food) has begun to act and it does this by letting any stimulus that has incidentally accompanied or anticipated the original stimulus act in its stead... In( instrumental conditioning) the organism selects from a large repertory of ... movements those of which the repetition is important with respect to the production of certain stimuli. The conditioned response ... does not prepare for the reinforcing stimulus, it produces it. The process is very probably that referred to in Thorndike's Law of Effect. (p. 111)

Classical conditioning arranges contingencies between stimuli and outcomes while instrumental conditioning arranges contingencies between responses and outcomes, and they together constitute the elements of associative learning.

In a typical classical conditioning experiment, the experimenter presents to the subject a stimulus known as a conditional stimulus, (CS), that is, a stimulus that at the beginning of the experiment does not bring about an unconditional response (UCR or UR). The CS is followed

closely in time, normally a few seconds, by the unconditional stimulus, (UCS or US), which is known to evoke an unconditional response. The experimenter records the subject's change in behaviour to the continued presentation of the CS. Classical conditioning is said to have occurred if the CS, after a series of CS-US pairings, comes to elicit a conditional response, (CR), which is similiar to the unconditional response elicited by unconditional stimulus presentation.

Pavlov (1927) reported his classical conditioning procedure as follows: a tone (CS) was presented to the subject (dog) a few seconds prior to the presentation of meat powder (US). The dog salivates (UCR) upon presentation of the meat powder (UCS) and after a series of CS-US pairings over a period of days the dog would salivate (CR) upon presentation of the tone (CS). The basic phenomena of classical conditioning include the following processes: acquisition, extinction, generalization, conditioned discrimination, higher-order conditioning, inhibition and spontaneous recovery (see Mackintosh, 1974; LoLordo, 1979; Kalish, 1981).

Instrumental or operant conditioning was developed from the experimental and theoretical foundations laid down by Thorndike (1911) and Skinner (1938). Whereas in classical conditioning the presentation of the unconditional stimulus is <u>independent</u> of the subject's behaviour, during instrumental conditioning it is <u>dependent</u> on the subject's behaviour. The defining feature of instrumental conditioning is that the UCS is delivered following the emission of the subject's operant response in a systematic manner. Take the example of a rat in a Skinner box. It is possible for the experimenter

to arrange for every lever press (CR) to be closely followed by food delivery (UCS). Food delivery is contingent upon lever pressing and the specific arrangement between food delivery and lever pressing defines the contingencies of reinforcement in operation for that particular subject at any given time (Ferster & Skinner, 1957). Whereas in classical conditioning, the CR is a specific response, in instrumental conditioning, the CR refers to a response class rather than one specific response in isolation. Given this distinction, instrumental conditioning then can be studied over a wider range of behaviours than classical conditioning, which gives instrumental conditioning greater generality in comparison to classical conditioning (Kazdin, 1978). The basic principles of instrumental conditioning include: reinforcement, extinction, punishment, stimulus control, contingencies of reinforcement, superstitious behaviour, and attention (see, Honig, 1966; Kimble, 1961; Honig & Staddon 1977; Fantino & Logan, 1979).

At the interface between classical and instrumental conditioning procedures is the avoidance learning procedure which has both classical and instrumental conditioning components. In the avoidance learning procedure CS-UCS pairings are presented to the subject (classical conditioning procedure) but they can be modified by the subject by performance of a pre-designated operant response (instrumental conditioning procedure). That is, if the operant response is performed after CS onset but before UCS onset, then an avoidance response is emitted which typically terminates the CS and prevents delivery of the UCS. Hoffman (1966) describes the discriminated avoidance procedure as follows:

> A neutral stimulus (CS) is scheduled to precede, and in this sense serves as a warning for, each occurrence of a noxious event. If the S emits an appropriate operant during the warning period (CS-UCS interval), the noxious event fails to occur. Under these conditions, discriminated avoidance represents a performance in which the S constantly prevents the noxious event, but seldom emits the operant in the absence of the warning signal. The behaviour is said to be discriminated in the sense that it is under the control of the stimulus. (p. 499)

One generally held view that cements the theoretical base of behaviour therapy and modification is that learning through principles of classical and instrumental conditioning plays an extremely important function in the development and maintenance of both adaptive and maladaptive behaviours (Kanfer & Phillips, 1970; Herrnstein, 1977; Kazdin, 1978; Rimm & Masters, 1979; Wolpe, 1982).

Eysenck (1960) argued:

> If the laws which have been formulated are, not necessarily true, but at least partially correct, then it must follow that we can make deductions from them to cover the type of behaviour represented by neurotic' patients, construct a model which will duplicate the important and relevant features of the patient and suggest new and possible helpful methods of treatment along lines laid down by learning theory. (p. 5).

Mowrer (1947, 1960) developed two-factor learning theory to explain avoidance learning behaviour investigated in the laboratory, and Stampfl & Levis (1967) developed Mowrer's theory to explain human neurotic behaviour.

Levis (1981) succinctly summarizes Mowrer's two-process theory of avoidance learning thus:

> The animal is learning two different responses. First of all, it is learning to become afraid of the tone CS, which can be conceived as a cue signalling the danger of being exposed to shock. The sequence of events by which fear is learned to the CS simply results from the pairing of the CS with the UCS. This type of learning is commonly referred to as classical conditioning. With repeated trials of the CS-UCS pairing, fear to the CS becomes strengthened. The second behaviour that the animal learns is how to protect itself from these events. This type of learning is referred to as instrumental or operant conditioning. Besides the aversive characteristics of conditioned fear, stimuli conditioned to the fear response are viewed as having motivational or drive properties that can heighten the subject's activity. This acquired drive will eventually result in the animal (performing the avoidance response). If this skeletal response is made contingent with CS offset ... the response will be reinforced by a reduction in the aversive stimulation ... Thus fear onset serves as a drive to activate avoidance responses, while fear reduction provides the condition necessary for reinforcement of the instrumental response. (pp. 336-351).

Two-process learning theory has been incorporated into both a theoretical account for the development and maintenance of human anxiety - fear-phobic reactions and their clinical treatment (Levis, 1979, 1980, 1981; Levis & Hare, 1977; Stampfl, 1966, 1970, 1976; Stampfl & Levis, 1967, 1969, 1973). This area of research will be more fully discussed later.

Another procedure with both classical and instrumental conditioning components is that termed conditioned suppression or conditioned emotional response (CER) (Estes & Skinner,

1941). In the Estes & Skinner (1941) procedure a subject's operant response rate is maintained by a schedule of positive reinforcement, the instrumental conditioning component. At intervals a tone (CS) is presented and at its offset an aversive stimulus is presented for a brief period of time, the classical conditioning component. After a series of CS-UCS pairings, the tone becomes a conditioned aversive stimulus as in classical fear conditioning. Typically upon presentation of the conditioned aversive stimulus the subject's autonomic nervous system activity changes (hence the term, conditioned emotional response) together with a concomitant decrease in operant response rate (hence the term, conditioned suppression), see (Davis, 1968; Lyon, 1968; Blackman, 1977; Davis & Hurwitz, 1977; Overmier & Lawry, 1979; Hoffman, 1969a, 1969b). Estes and Skinner (1941) suggested the CER procedure may be appropriate for the quantitative study of anxiety, that is, the amount of suppression of operant responding during CS presentation could be an appropriate index of anxiety. Subsequent experimentation has failed to completely corroborate Estes and Skinner's assertion as Davis (1979) states:

> Although literally hundreds of experiments have convinced even the most skeptical observer that conditioned suppression "works", the question of <u>how</u> or <u>why</u> still remains a mystery. Although proclamation about motivational or response incompatibility are often made ... no one has elucidated the exact manner in which an aversive Pavlovian CS-US produces changes in the rate of an appetitive operant baseline. (pp. 198-199.)

While no one theoretical account of the complex phenomenon of conditioned suppression has been dominant, the

procedure itself has been most illuminating in a number of research areas as Blackman (1977) illustrates:

> ... The procedure has proved to be successful in providing a sensitive dependent variable for the study of the necessary and sufficient conditions for the development of an acquired reflex ... provided empirical evidence which has been related to motivational theories of behavior and the role of classical conditioning in motivation, to the study of emotion, to the relations between physiological events and overt behavior,to the study of the effects of potential anxiolytic agents, and to many other important problems. (p. 360).

At approximately the same time the avoidance learning paradigm was developed there was the emergence of another field of scientific endeavour called behavioural pharmacology or psychopharmacology (Thompson & Schuster, 1968; Iversen & Iversen, 1975; Glick & Goldfarb, 1976; Blackman & Sanger, 1978). Thompson & Schuster (1968) have defined behavioural pharmacology as:

> ... a branch of biological science that used the tools and concepts of experimental psychology and pharmacology to explore the behavioural actions of drugs. (p.1).

While the use of drugs to alter psychological functioning and/or physical health has been with mankind since the time of antiquity, behavioural pharmacology as an experimental science has a much shorter history. Skinner and Heron's (1937) paper represents an early drug study investigation, but the major thrust of experimentation began in the 1950's with the work of Dews (1955, 1956). From this date research mainly focused on the use of instrumental conditioning techinques to elucidate drug effects on behaviour. Sanger

(1981) states the advantages of instrumental conditioning techniques in behavioural pharmacology research are:

1. Allows a fine control of behaviour with important variables identified.
2. Similiar behavioural baselines can be obtained in a variety of species.
3. Allows an analysis of drug effects on ongoing behaviour.
4. Provides baselines very sensitive to the actions of many drugs. (p. 235).

Thompson & Boren (1977) reiterate the above sentiments by saying:

> ... that operant behavioural pharmacology has, by and large, succeeded in satisfying the two major requisites of a scientific domain concerned with the analysis of drug actions on behaviour: (1) The provision of sensitive and reliable behavioural procedures; and (2) the provision of an objective, operationally based conceptual framework within which to interpret the results of experiments on the behavioural actions of drugs. (p. 541).

A number of procedures have been used to examine drug induced performance changes in animal subjects, for example, multiple schedules (Hearst, 1960; Herrnstein, 1958; Waller, 1961; Dews, 1971); titration or adjusting procedures for the detection of sensory thresholds (Blough, 1957a; 1958; Weiss & Laties, 1959, 1963); stimulus generalization tests (Hearst, 1964;Dykstra & Appel, 1970, 1972); and discrete trial procedures (Heise, 1975; McFarlain, 1973; Blough, 1957b; Berryman, Jarvik & Nevin, 1962). While each of these procedures has advantages, as outlined earlier, they also have disadvantages for the study of changes in performance produced by drug action. With the above procedures the dependent variable is rate or probability of responding on

an operant task, then it is possible that the drug effect on indices of discriminability maybe confounded with a possible drug rate-dependent effect (Kelleher & Morse, 1968; Sanger & Blackman, 1976). That is, instrumental conditioning procedures do not always allow separation of effects on stimulus input (stimulus discriminability) from effects on response output (response bias) leading to the possibility of drawing erroneous conclusions regarding drug action.

One solution to the dilemnia is to use a theoretical and procedural approach originally developed to specify the electronic detection of radar signals in noise called signal detectability or statistical decision theory (SDT), (Green & Swets, 1966; Swets, 1973). This approach does allow for the separate analysis of changes in performance due to variation in sensitivity to sensory cues (stimulus input) from changes in performance due to variations in response rate (response output). This approach has now been applied to a variety of experimental situations with animal subjects including matching performance (Davison & Tustin, 1978; McCarthy, 1981, 1983; McCarthy & Davison, 1979; McCarthy, Davison & Jenkins, 1982; Nevin, Jenkins & Yarensky, 1982; Logue, 1983); and drug-induced behaviour change research (Warburton & Brown, 1971, 1972; Dykstra & Appel, 1974; Appel & Dykstra, 1977; Francis & Cooper, 1979).

# CHAPTER ONE

# INTRODUCTION

## Historical Perspectives to the study of Avoidance Learning Behaviour

### The Russian Influence

Ivan M. Sechenov is generally believed to be the founder of Russian Physiology (Sechenov, 1865/1965). Early in his career his experimental focus was on the inhibition of reflex movements by the cerebral cortex. This lead him to show that there was reason to believe a physiological basis for psychological processes. Sechenov's main thesis was thus: psychological activity can be explained by reflex activity. Sechenov believed behaviour was the joint function of learning and environment events, and in doing so, mirrored the theoretical developments that were later to take place in America with J.B. Watson and B.F. Skinner. It was Sechenov who laid the theoretical foundation which Pavlov followed in his investigations.

Pavlov initially investigated reflexes associated with glandular secretions (Pavlov, 1902) then to the investigation of conditioned reflexes. Pavlov viewed conditioning as a function of cortical extinction and cortical inhibition (Pavlov, 1927). Kazdin (1982) succintly summarized Pavlov's main contribution to psychology as

> ... objectively investigating conditioned reflexes from the standpoint of a physiologist. He strongly advocated objectivism in research and was critical of subjective lines of psychological inquiry (eg., Pavlov, 1906). (p. 7.)

Pavlov will be encountered again shortly, but let us turn to a contemporary of Pavlov's who it can be said performed the first avoidance experiment as we know it today.

Whereas Pavlov had concentrated on reflexes of the glands and the digestive system, Bechterev's research focused on reflexes of the motor system, (Becheterev, 1932). Becheterev's use of shock as the UCS and muscle flexion as the UCR allowed greater flexibility from the experimental viewpoint than Pavlov's classical conditioning method, and was also more applicable to human behaviour analysis (Bechterev, 1913). Research conducted by Bechterev culminated in the development of a discipline he termed "reflexology" (Bechterev, 1932) which focused on explanations of human personality, adaptive and maladaptive behaviour in terms of conditioned reflexes. But more importantly the first avoidance learning experiment was conducted in Bechterev's laboratory, although at the time the experimenters thought it to be a classical conditioning experiment (Hilgard & Marquis, 1940; Bolles, 1972b).

The first avoidance learning experiment was conducted by Molotkov (1910; cited in Razran 1956), using humans as subjects. The procedure was described by Bolles (1972b) as follows:

> A human S's finger is placed on a metal electrode: after a 2-second conditioned stimulus (CS) ... electricity from an inductorium is applied. The unconditioned response (UR) is finger withdrawal. After just a few trials, the finger also withdraws to the CS, prior to the onset of shock. Because the CS comes to elicit the response originally elicited by the US, the learning looks like classical

> conditioning. But look again. The finger is merely placed on the electrode so that any movement will break the shock circuit. The S is avoiding shock by responding to the CS. Thus, the learning looks like instrumental avoidance learning. (p. 98).

Starytzin (1926; cited in Razran 1956), investigated avoidance learning in dogs and Starytzin's procedure was later refined and modified by Petropavlovsky (1934; cited in Razran 1956). The Russian researchers had not conceptualized their avoidance learning procedure to represent a different form of conditioning from their classical conditioning procedure. The conceptual distinction was later made by American researchers (Schlosberg, 1934, 1937; Skinner, 1935 1937; Brogden, Lipman & Culler, 1938; Mowrer, 1939).

Thus, in Bechterev's laboratory the experimental procedure permitted the subject to escape/avoid the aversive US, providing the necessary procedural requirements for the graduation from classical conditioning (no escape/avoidance of the US possible) to escape/avoidance learning.

## The American Influence

The first reported avoidance procedure from an American laboratory was that conducted by Hamel (1919). It was a finger withdrawal experiment in the tradition of Bechterev. Carr and Freeman (1919) and Yarbrough (1921) also studied avoidance learning but their procedure was confounded by punishment contingencies for incorrect responding. Non human animal subjects were first used in avoidance experiments by Upton (1929), Wever (1930) and Culler, Finch, Girden and Brogden (1935).

Warner (1932a), using rats as subjects, was the first

experimenter to include an escape contingency into the hitherto typical avoidance learning procedure. Before Warner, UCS offset was independent of the subject's behaviour, it could be avoided during the CS-UCS interval, but not escaped after UCS onset. Warner allowed the subject to terminate the UCS by performing the CR, in this case, jumping over a hurdle separating two chambers of a shuttlebox. As stated earlier, at the time of Warner's experiment the conceptual distinction between classical conditioning and avoidance learning had yet to be made, with Warner discussing his experiment totally within a classical conditioning framework. Warner's (1932a) procedure was the prototype of much discrete-trial avoidance-escape learning research to follow.

Warner (1932b) reported a second experiment in which he noted response topography between avoidance and escape responses was different. Presentation of the UCS changed an orienting response to a response functionally similar to the UCR. Warner's experiments set the scene for a series of experiments by Schlosberg (Schlosberg, 1934, 1936; Kappauf & Schlosberg, 1937) who was the first researcher to directly compare classical conditioning and avoidance learning procedures. Schlosberg reported no difference between the procedures, but this conclusion was unwarranted given he failed to obtain sustainable conditioning of tail withdrawal or leg flexion responses in the rat, although such procedures with dogs and pigs had demonstrated good conditioning (Liddell, 1934). While empirically Schlosberg's results were disappointing, conceptually he made the leap in distinguishing and comparing two conditioning procedures.

A number of comparison experiments, classical

conditioning vs avoidance learning, followed Schlosberg's research. They can be grouped into those studies that found no differences in performance between the two procedures (Munn, 1939; Hilden, 1937; Whatmore, Morgan & Kleitman, 1946; and Wickens & Platt, 1954) and those that showed superior avoidance learning performance (Hunter, 1935; Brogden, Lipman & Culler, 1938; Sheffield, 1948; Traum & Horton, cited in Mowrer, 1950; and Gibson, 1952).

Solomon & Brush (1956) succinctly summarized this period of research by saying:

> We are led to conclude from the experimental studies concerning the relative effectiveness of classical aversive conditioning and avoidance training procedures that the latter produces better and more stable aversive motor behaviour, while the former produces more anxiety or emotion. (p. 236).

From this period onwards, research focused on two inter-related aspects of the avoidance learning procedure, varying parameters considered to have a significant effect on conditioned avoidance learning and developing theory to explain avoidance learning. Thus, the research emphasis shifted from comparison studies to research guided by the following questions:

1. why does avoidance responding persist in the absence of UCS presentation, contrary to the classical conditioning situation?
2. What are the mechanisms and experimental contingencies that establish avoidance learning behaviour?
3. What maintains and motivates avoidance learning?
4. What role does fear play in avoidance learning and how is is learned?

5. Why does avoidance learning performance vary across different CRs?
6. Is avoidance learning especially resistant to extinction? and
7. What procedures facilitate extinction of avoidance responding?

Theories of avoidance learning were developed in an attempt to provide coherent answers to some of these questions.

The next section examines these theories as they were developed to explain the acquisition, maintenance and extinction of conditioned avoidance learning.

## Theoretical Accounts of Conditioned Avoidance Learning: Acquisition processes

### Two-Process Fear Mediation Theory

Two-process avoidance theory was proposed to explain how avoidance responding is acquired and maintained. Mowrer's theory was developed using a discrete-trial discriminated avoidance procedure such as running in a shuttlebox. Mowrer's (1947) 2-process theory was briefly outlined earlier, but to recapitulate, Mowrer stated that after a series of CS - aversive UCS pairings, the CS comes to elicit the conditioned response of fear by the process of classical conditioning. Conditioned fear is also said to motivate the avoidance response, an instrumental response, which terminates the CS and by preventing exposure to the aversive UCS is reinforced by fear reduction. In Mowrer's theory fear has drive properties, which motivates the subject to perform the conditioned avoidance response, but once fear has extinguished

so should avoidance responding.

A number of animal studies have supported

1. the motivational effects of conditioned fear (Amsel & Maltzman, 1950; Brown, Kalish & Farber, 1951),
2. fear reduction acting as a reinforcer for avoidance responding (Mowrer, 1947, 1950; Brown & Jacobs, 1949; Miller, 1948),
3. extinction of fear and avoidance behaviour with CS presentation in the absence of the UCS (Baum, 1970; Black, 1958; Weinberger, 1965).

Although Mowrer (1960) revised the 1947 version of his 2-process theory of avoidance behaviour to include appetitive behaviour, Levis (1982) states:

> Mowrer's 1947 version of avoidance behaviour, however still seems to be the preferred interpretation (see Rescorla & Solomon, 1967) ... Although not free from criticism (Herrnstein, 1969), the two-factor theory has survived the test of time and is still considered a very viable explanatory model for infrahuman and human avoidance behaviour. (p. 69).

While Mowrer's 2-process theory explained how reinforcement occurred on non-UCS exposure trials (avoidance trials), other theorists believed the role of fear as a drive whose reduction was reinforcing as unnecessary (Schoenfeld, 1950; Dinsmoor, 1954; Anger, 1963).

## Two-Process Aversion Theory

Schoenfeld (1950) maintained that during avoidance training the CS paired with shock became aversive as a secondary aversive stimulus, with consequent CS termination reinforcing the avoidance CR. While Mowrer (1947) uses

"fear" as an hypothetical construct, a Pavlovian emotional state (MacCorquodale & Meehl, 1948), Schoenfeld uses "aversive" to indicate that termination of the CS increases the probability of the avoidance CR occurring on subsequent trials. Schoenfeld (1950) states the position of the CS in avoidance conditioning as follows:

> The avoidance response, by this formulation, is not really avoidance at all, or at least is only incidentally so. Its function is not to avoid, and it is not made "in order to avoid". Rather, it is primarily an escape response, reinforced by the termination of secondary noxious stimuli, including proprioceptive and tactile ones, and possibly also reinforced by the production of proprioceptive secondary positive reinforcers. (p. 88).

A number of studies have provided support for the Schoenfeld position (Mowrer & Lamoreaux, 1942; Kamin, 1954, 1956) although both Meyer, Cho & Wesemann (1960) and Masterton (1970) reported CS termination failed to reinforce avoidance responding.

Sidman (1953a, b) reported a new avoidance procedure in which the subject could avoid the UCS indefinitely although no explicit CS is ever presented. This avoidance procedure is variously termed Sidman, nondiscriminated or free-operant avoidance. In its original form, it involves the presentation of brief inescapable shocks, in the absence of responding, with a fixed time period (shock - shock interval, S-S) between shocks. If during the S-S interval the subject responds this had the effect of switching the procedure to the response - shock interval, R-S interval. Normally the R-S interval is of a longer duration than the S-S interval. Responding postpones UCS delivery and spaced continuous

responding can indefinitely postpone shock, ie., avoidance performance. A number of variations to this original Sidman avoidance procedure have been developed and investigated (Hineline, 1977; Sidman, 1966).

Avoidance performance in the absence of an explicit CS presents difficulties for both the 2-process fear theory and 2-process aversion theory. How did these theories explain Sidman avoidance responding? The answer was to "invent" conditioned stimuli which acted with the properties of explicit CSs of the discriminated discrete-trial avoidance procedures. Schoenfeld (1950) states that feedback from the animal's behaviour becomes the CS. Inappropriate behaviours will be paired with shock and become aversive, but avoidance behaviour terminates aversive proprioceptive feedback and is therefore reinforced. Anger (1963) developed the concept of conditioned aversive temporal stimuli (CATS) to explain avoidance responding of the Sidman avoidance procedure. While Anger like Schoenfeld believed the avoidance response terminated aversive proprioceptive feedback, Anger extended the analysis by arguing the subject developed a sense of time between shock presentations and it was this shock interval itself that acted as the CS. As time since the last shock presentation increases, there is a concomittant increase in the aversiveness of the associated stimuli which leads to avoidance responding, eliminating the aversive stimuli.

As researchers focused attention on avoidance learning a number of "puzzles and paradoxes" (Masterson & Crawford, 1982) developed which required theoretical resolution. This lead to both modifications to the two-process fear mediation theory and development of new theories to account for

avoidance learning.

Anxiety Conservation and the Partial Irreversibility Principles

Solomon and colleagues (Brush, Brush & Solomon, 1955; Solomon, Kamin & Wynne, 1953; Solomon & Wynne, 1953, 1954) performed a series of experiments on traumatic avoidance learning with dogs as subjects. In the Solomon, Kamin and Wynne (1953), and Solomon and Wynne (1953) studies dogs learned the shuttlebox hurdle avoidance response very rapidly but showed a high resistance to extinction. This result presents a problem for two-process theory as Mineka (1979) illustrates:

> The dilemna for two process theory is that after dozens or hundreds of consecutive avoidance responses, the source of reinforcement for responding is no longer apparent because each successful avoidance trial constitutes a Pavlavian extinction trial. Hence the fear CR should gradually extinguish, thus removing CS termination as a possible source of reinforcement. After that, the avoidance operant should proceed to extinguish. (pp. 989-990).

To explain high resistance to extinction Solomon & Wynne (1954) developed the principles of anxiety conservation and partial irreversibility of the CR learned with traumatic shock. They ascertained that short CS - exposures from short avoidance latency responses do not permit enough time for any reduction to occur in the classical conditioned fear reaction. On these trials, fear reduction fails to occur and the avoidance response is consequently weakened. This weakening effect in turn leads to longer latency avoidance

responses which elicit the conditioned fear reaction. On longer latency avoidance trials the avoidance response is strengthened and reinforced. This responding in turn generates shorter response latencies again as the fear reaction to longer CS exposures secondarily conditions fear to shorter CS exposures.

The anxiety conservation principle was found to be unsatisfactory because two process theory requires fear to be present to elicit avoidance responding. If on short - CS exposure trials, classically conditioned fear is absent as postulated by Solomon & Wynne (1954) then how can the avoidance response occur? What motivates responding?

Stampfl (1960) addressing himself to this question noted that the studies of Brush (1957); Brush, Brush & Solomon (1955); Solomon, Kamin & Wynne (1953); and Solomon & Wynne (1953) all reported extreme resistance to extinction while the studies of Brush, E. (1957); Denny, Koons & Mason (1959); and Kamin (1954) reported relatively rapid extinction. Stampfl looking closely at the procedures employed by all the above studies found all the former studies finding resistance to extinction had used a drop-gate to separate the shuttlebox compartments while the drop-gate was absent in the studies reporting rapid extinction. Stampfl proposed that during avoidance conditioning of humans many stimuli are present and take the form of a conditioned emotional reaction. The stimuli, he argues, are normally arranged in serial order along a temporal dimension during avoidance conditioning acquisition. Human avoidance responses occur early in the sequence of serial CSs preventing exposure to other CSs in the chain and thereby prolonging the extinction process.

After repeated exposure to early CSs, extinction to these CSs occurs, thus exposing the person to CSs later in the chain which then elicit classically conditioned fear, and at the same time secondarily reinforcing those CSs earlier in the stimulus sequence. These early CSs regain the potential to elicit fear which, in turn, motivates avoidance responding and so the process continues.

On the basis of the above analysis of human avoidance responding, Stampfl (1960) argues that the drop-gate procedure increased the CS complexity and thus produced extreme resistance to extinction, an assertion supported by Levis (1971) and noted by Church, Brush & Solomon (1956). Stampfl then argued if short-latency avoidance responses conserved fear to longer-latency CRs then dividing the CS-UCS interval into discriminable stimulus segments ordered sequentially should maximize the process of conservation. The prediction that serial-conditioned stimuli presentation will produce resistance to extinction compared with the non-serial CS procedure has been verified by Kostaneck & Sawrey (1965), Levis (1971), Levis & Stampfl (1972), Levis & Boyd (1979).

Like Solomon and Wynne (1954), Stampfl (1960) maintained that the fear level associated with a particular part the serially divided CS-UCS interval decreases as the segment's temporal distance from the end of the CS-UCS interval increases. This prediction has also been supported by the following avoidance learning studies Dubin & Levis (1973), Levis and Dubin (1973), Levis and Stampfl (1972) and Boyd and Levis (1976).

The critical difference between the Solomon and Wynne (1954) and Stampfl (1960) positions is exemplified by

Levis (1981):

> However, unlike the Solomon and Wynne interpretation, Stampfl argues that the fear response is elicited by the CS when short-latency avoidance responses occurs ... At an asymptotic state, little reinforcement (fear reduction) is needed to elicit responding. Short CS exposure is then viewed as eliciting a fractional anticipatory fear response which at an asymptotic response-level is capable of motivating avoidance responding for some time. (p. 360).

To explain extreme resistance to extinction found in their research, Solomon and Wynne (1954) also proposed the principle of partial irreversibility. They ascertained that a traumatic fear reaction to conditioned stimuli produces a permanent increase in the probability of an occurrence of the fear reaction in the presence of the CS pattern. Research has failed to support the partial irreversibility principle (Brush, F. 1957; Levis, 1966a, b; Maatsch, 1959).

It was from this experimental background that Stampfl & Levis developed the technique of Implosive Therapy for the treatment of fear motivated human neurotic behaviours (Stampfl, 1970; Stampfl & Levis, 1967a, b) which will be examined more fully in the section on methods of facilitating extinction of avoidance performance.

## Effective Reinforcement Theory

Mowrer's two process theory was further modified in response to experimental data which seemed incompatible with the theory's predictions. The experimental data concerned the comparison of one-way versus two-way avoidance learning performance. The standard one-way avoidance task requires the subject to shuttle (ie., move from one chamber, designated

the start chamber, to a distinctly different adjacent chamber, the goal chamber) in order to avoid/escape the shock UCS. The intertrial interval, in a discrete trial procedure, is normally spent in the goal chamber and immediately prior to the start of the next trial the subject is placed into the start chamber. The avoidance response is normally learned in a few trials. The two-way avoidance response task differs from the one-way task in that, instead of being placed in the start chamber before the commencement of every trial, the subject remains in the chamber in which it spent the intertrial interval. The subject must shuttle back to the chamber it had just come from when the CS is presented. Normally in the two-way task both chambers are identical, with the UCS being presented equally often in each chamber, while for the one-way task the UCS is only presented in the start chamber. One-way avoidance performance is directly related to shock intensity (Dieter, 1976; Moyer & Korn, 1966) but the two-way avoidance performance is inversely related to shock intensity (Anisman & Waller, 1972; McAllister, McAllister & Dieter, 1976; Moyer & Korn, 1964; Theios, Lynch & Lowe, 1966). The finding that two-way avoidance performance deteriorates as shock intensity increases presents a difficulty for Mowrer's 2-process theory. According to 2-process theory, as shock intensity increases, the fear elicited by the CS should also increase with a concomittant increase in fear reduction occurring with CS termination and reinforcement for the avoidance response.

Effective reinforcement theory (McAllister, McAllister & Dieter, 1976; McAllister, McAllister, Dieter & James, 1979) was proposed as an extension to Mowrer's 2-process theory to

account for the relationship between shock intensity and 2-way avoidance performance. This theory states that avoidance performance is determined by both the amount of fear reduction occurring upon CS termination and fear level persent after performance of the avoidance response. As with Mowrer (1947), fear reduction is positively related to avoidance performance, but adds, the amount of fear following an avoidance response is negatively related to performance. Effective reinforcement for avoidance responding is then a joint function of fear reduction associated with CS termination and the amount of fear remaining after completion of the avoidance response. Effective reinforcement theory also states that if both fear reduction and the amount of fear remaining after an avoidance response increase equally, the deleterious effect of the fear remaining after avoidance responding increases faster than the facilitating effect of fear reduction, ie., avoidance performance will deteriorate. McAllister, McAllister, Dieter & James (1979) explain 2-way avoidance responding according to effective reinforcement theory as follows:

> In the signaled two-way avoidance task, fear becomes conditioned to both the discrete conditioned stimulus (CS) and to the situational cues of each shuttle compartment when shock occurs on escape trials, with more fear being conditioned to these stimuli the more intense the shock. Although the amount of fear reduction would be greater in a strong - than in a weak-shock group when the CS is terminated following an avoidance response, this advantage is more than offset by the greater amount of fear of situational cues present following the response. Thus with strong shock, there would be

> less effective reinforcement, and hence, avoidance performance would be inferior. (p. 165).

McAllister et al. similarly explain one-way avoidance acquisition as follows:

> In contrast, in a one-way avoidance task, where the response leads to a distinctive safe area, the amount of fear of situational cues following the response would be zero or minimal. Under this circumstance, effective reinforcement would be determined simply by the amount of fear reduction occurring with the response. Therefore, avoidance performance should be superior with strong shock. (pp.165-166).

Effective reinforcement theory's explaination of 2-way avoidance acquisition has been supported by Dieter (1977) and McAllister et al. (1979) who reported that the deleterious effect of strong shock on avoidance performance can be eliminated by reducing fear conditioned to situational cues. Eliminating fear to situational cues, with shock intensity constant, also facilitates 2-way avoidance performance as predicted by effective reinforcement theory (Boyd & Levis, 1979; McAllister et al., 1979; Modaresi, 1975). Increasing fear to situational cues with escape trials decreases 2-way avoidance performance (Bloom & Campbell, 1966; Mowrer, 1940).

In summary, Mowrer's (1947) 2-process theory has undergone a number of revisions since Mowrer first proposed it to explain avoidance learning. While it has stood the test of time exceedingly well, a number of theorists have adopted an entirely different approach to explain the "puzzles and paradoxes" of avoidance learning performance.

Contiguity Theory

Contiguity theory espoused by Guthrie (1935) has been applied to avoidance by Hull (1929), Sheffield (1948) and Sheffield & Temmer (1950). Contiguity theory is basically a Pavlovian view of avoidance learning, that is, learning via a contiguous relationship between the CS and UCS with no reference to a reinforcement mechanism. Contiguity theory states the aversive UCS elicits the avoidance response and this elicitation during CS presentation results in the CS acquiring the power to elicit the avoidance response through classical conditioning. It is also argued that the avoidance contingency is incidental to the emergence of the avoidance response. Contiguity theory provides a number of testable predictions which include:

1. only responses elicited by the UCS should get learned as avoidance responses;
2. the avoidance contingency is irrelevant;
3. when the subject is avoiding on every trial this constitutes a classical extinction procedure.

Research has failed to support the predictions derived from continguity theory. Responses other than those elicited by the UCS have been shown to act a avoidance responses (Fonberg, 1962; DiCara & Miller, 1968). If the avoidance contingency is irrelevant then classical conditioning should reliably lead to better performance than avoidance contingencies. As has been stated previously, the avoidance procedure is normally superior to classical conditioning (Brogden, Lipman & Culler, 1938; Wahlsten & Cole, 1972; Woodard & Bitterman, 1973). Finally a number of studies

have reported persistence of avoidance responding in the absence of UCS presentation contrary to contiguity theory (Solomon & Wynne, 1953; Solomon, Kamin & Wynne, 1953).

Reinforcement Theory

Hull (1943) presented a reinforcement analysis of avoidance learning. Hull maintained avoidance responding was reinforced by aversive UCS termination. Response latency is inversely related to his concept of habit strength (sHr) with avoidance responses being elicited anticipatorily by the stimulus conditions. Again, as with contiguity theory, only responses occurring during UCS presentation will be learned as avoidance responses with the avoidance contingency being irrelevant to this learning but the escape contingency being critical. Hull therefore regarded avoidance responses as emerging from escape responding or in other words as anticipatory escape responses.

Bolles, Stokes and Younger (1966) reported the effect of the contingency is determined primarily by the response task under investigation, for example, shuttlebox or wheel running. Hurwitz (1964a) found the escape contingency interfered with avoidance responding, another result inconsistent with Hullian reinforcement theory. The evidence, thus suggests that reinforcement theory is not a valid account of avoidance learning.

Relaxation Theory

The concept of relaxation to explain avoidance responding can be found in elicitation theory (Denny, 1966, 1967; Denny & Adelman, 1955), the elicitation hypothesis (Bolles,

1967; Maatsch, 1954) and relaxation theory (Denny, 1971). Denny (1971) extended the function of CS termination providing fear-reduction (Mowrer, 1947) to also include CS termination providing the positive consequences of relief and relaxation. Relaxation behaviours Denny considered to by antagonistic to fear-motivated avoidance responses. According to Denny's (1971) relaxation theory the subject performs the avoidance response to achieve the post UCS termination situation of relief and relaxation. For Mowrer (1947) reinforcement for avoidance responding was provided by fear reduction through CS-termination, for Denny (1971) reinforcement for avoidance responding is provided by relief and relaxation which is more desirable than being in the presence of the CS and UCS. While Denny (1971) reviews a number of studies supporting relaxation, the theory has difficulties explaining free-operant avoidance responding, rapid acquisition of one-way avoidance when the inter-trial interval is spent in the shock compartment (Masterson, Crawford & Bartter, 1978) and response prevention or flooding - facilitated extinction of avoidance behaviour.

Conditioned Inhibition of Fear Theory

Konorski (1948) and Soltysik (1963) expanded Mowrer's (1947) two-process theory to account for the resistance to extinction data (e.g., Solomon, Kamin & Wynne, 1953) by postulating that the avoidance response becomes a conditioned inhibitor ($CS^-$) for shock.

A Pavlovian conditioned inhibitor is a stimulus paired with the CS in the absence of the UCS resulting in the CR ceasing to be elicited by the $CS^-$ - CS pairing, but being

elicited by CS alone presentations. Extrapolating from Pavlovian conditioned inhibition research to the avoidance paradigm, Soltysik argues that in avoidance the CS is paired with stimuli produced by the avoidance response that predict no shock. These stimuli come to be conditioned inhibitors of fear. It is this inhibition of fear that provides reinforcement for avoidance responding. Also, the $CS^-$ stimuli "protect" the avoidance response from extinction (Bull & Overmier, 1968; Morris, 1974b; Rescorla, 1968). One difficulty with this extrapolation is that during Pavlovian conditioning the $CS^-$ precedes the CS whereas in avoidance conditioning the CS precedes the $CS^-$. Soltysik (1960) modified the Pavlovian salivary conditioning procedure so that the temporal relationship between CS and $CS^-$ was the same as that in avoidance conditioning. Soltysik reported an intact salivary response after the presentation of a series of CS - $CS^-$ pairings, thus confirming his expectations. Using the avoidance paradigm to directly test Soltysik's theory, LoLordo & Rescorla (1966) and Johnston, Clayton and Seligman (1972) cited in Seligman and Johnston (1973), reported results inconsistent with Soltysik's theory. Also, several studies have reported that fear, when measured using the conditioned emotional response (CER) procedure, is not as great during later stages of avoidance acquisition as in early avoidance trials, thus indicating a dissociation between fear and prolonged avoidance performance (Kamin, Brimer & Black, 1963; Mineka & Gino, 1979b, 1980; Mineka, Miller, Gino & Gienche, 1981; Starr & Mineka, 1977). Alteration of fear over the duration of avoidance training presents a difficulty for Soltysik's theory.

Safety-Signal Theory

Bolles (1970, 1972), Weisman and Litner (1969, 1972) and Gray (1971) proposed a safety-signal account of avoidance learning similar to that of Soltysik (1963). Mineka (1979) summarizes this theory as follows:

> (it) assumes that the avoidance response becomes a $CS^-$ for shock ... However, this theory does not require that the $CS^-$ protect the $CS^+$ for extinction, because the $CS^-$ assumes the role of a positive reinforcer. The animal continues to make avoidance responses because the response itself ($CS^-$) becomes a positive reinforcer and so fear is not necessary to continue to motivate the avoidance response once the response has become a good $CS^-$. (p. 990).

Thus, the avoidance response itself becomes a safety-signal for the absence of shock and the safety-signal acts as a positive reinforcer. Avoidance responding is facilitated by the introduction of feedback stimuli (Bolles & Grossen, 1969; D'Amato, Fazzaro & Etkin, 1968) as predicted by safety-signal theory.

A difficulty for safety-signal theory is asymptotic avoidance responding, i.e., how do stimuli paired with the absence of shock on many trials retain their positively reinforcing properties? In the absence of UCS presentation, it is yet to be shown that the $CS^-$ have positively reinforcing properties (LoLordo, 1969; Seligman and Johnston, 1973). Further research is required to ascertain the role of safety-signals during asymptotic avoidance responding. However, it has been demonstrated that both CS termination and $CS^-$ as safety-signals both contribute to strengthening avoidance responding (Cicala & Owen, 1976; Owen, Cicala & Herdegen,

1978).

While there is supporting evidence for both contitioned inhibition of fear and safety-signal theories they both have problems in adequately explaining extinction and response prevention effects on avoidance behaviour, which will be discussed in the next section.

Shock frequency reduction theory

A major alternative theoretical account of avoidance learning to two-process theory and its modifications is shock frequency reduction theory (de Villiers, 1974; Herrnstein, 1969; Herrnstein & Hineline, 1966; Sidman, 1962; Hineline, 1977; D'Amato, 1970).

In an avoidance session of length T minutes, X shocks (UCS) are programmed to occur in the absence of effective avoidance responding. If Y number of avoidance responses are emitted, then X - Y shocks are received by the subject. Shock frequency in the absence of avoidance responding is then X/T, and in the presence of avoidance responding, X-Y/T, thus indicating shock frequency is reduced in the presence of responding in comparison to the absence of effective avoidance responding. It is this shock frequency reduction that Herrnstein & Hineline (1966), Herrnstein (1969), Hineline (1977) and others propose as the reinforcer for avoidance responding, not fear reduction as proposed by two-process theory. Herrnstein et al. argue that the warning signal acts as a discriminative stimulus, $S^D$, which signals the avoidance response will be reinforced. Shock frequency reduction theory focuses on the avoidance of the UCS as being critical to the motivation of avoidance responding

while safety-signal theory focuses on stimuli correlated with the absence of the UCS rather than its absence per se.

Supportive evidence for shock frequency reduction theory comes from the number of experiments which have shown the warning signal acting as a discriminative stimulus rather than a CS, a motivational mediator for avoidance responding (D'Amato, Fazzaro & Etkin, 1968; D'Amato, 1970; Sidman, 1955, 1957; Field & Boren, 1963, see Hineline, 1977 for a review). Further support for this theory has come from studies indicating that avoidance of the UCS rather than CS termination can reinforce avoidance responding (Kamin, 1956, 1957; Bolles, Stokes & Younger, 1966) and the maintenance of avoidance responding by contingencies which vary shock frequency (de Villiers, 1972, 1974; Herrnstein & Hineline, 1966; Hineline, 1977).

While there has been strong support for shock-frequency reduction theory in the operant conditioning literature (Hineline, 1977; Fantino & Logan, 1979), it does have some problems. Gardner & Lewis (1976) and Hineline (1970) reported avoidance responding when such responding failed to produce shock frequency reduction as would be expected, indeed, considered to be necessary, by shock frequency reduction theory. Masterson & Crawford (1982) criticise shock frequency reduction theory on theoretical grounds as follows:

> ... despite its descriptive stance, it implies the existence of an expectancy learning process ... The crucial point is that the nonoccurrence of shock (or the occurrence of a lower rate of shock) can only be reinforcing <u>in contrast</u> to an expectation that shock will occur (or that shock

> will occur at a higher rate) ... What are the rules governing the acquisition of expectancies? How do expectancies govern behaviour? Unfortunately, this theoretical path is not a smooth one. (p. 672).

Species Specific defense reaction (SSDR) theory

Bolles (1970, 1971, 1972, 1975, 1978) developed SSDR theory primarily to resolve the "response problem", Bolles (1970), that had plagued theorists studying avoidance acquisition. The response problem refers to the finding in the avoidance learning literature that some responses seem to be acquired more rapidly as conditioned avoidance responses than others, which has been termed a "continuum of difficulty" (Crawford & Masterson, 1982). Responses such as one-way avoidance and jump-up acquisition are learned rapidly (Maatsch, 1959; Theios, 1963; Theios, Lynch & Lowe, 1966). Other responses, intermediate along this continuum, are learned less rapidly, such as shuttle box and wheel running acquisition (Theios & Dunaway, 1964; Bolles, Stokes & Younger, 1966). Lever presses, chain pulling and wheel turning avoidance responses are learned very slowly, if at all (Meyer, Cho & Wesemann, 1960; D'Amato & Schiff, 1964; Chapman & Bolles, 1964; Pearl & Edwards, 1962; Masterson, 1970). Also parametric manipulations have been found to effect avoidance learning of one type of response but not another (Bolles & Seelbach, 1964; Kamin, 1956; Bolles, 1969).

It is difficult to train pigeons to key-push in order to avoid shock (Hineline & Rachlin, 1969; Hoffman & Flesher, 1959; Rachlin, 1969; Rachlin & Hineline, 1967). Whereas they can be trained to locomote (MacPhail, 1968), flap their wings (Rachlin, 1969) and fly (Bedford & Anger, 1968) in

order to avoid shock.

Bolles (1970) states that fear, elicited through classical conditioning contingencies, limits the subject's behavioural repertoire to a set of SSDRs - freezing, fleeing and fighting. If the designated conditioned avoidance response involves freezing, fleeing or fighting then it will be learned rapidly, if it is not, then learning would be slow. Bolles has modified the SSDR theory over the years and these modifications are discussed in detail by Crawford & Masterson (1982), who in summary of SSDR theory development state:

> ... the SSDR theory presented by Bolles (1970) and later modified (Bolles 1971, 1972a, 1972b, 1975, 1978) has remained constant in its assumption that fear dramatically limits the rat's response repertoire. It has increasingly specified the motivational processes presumed to be operating, culminating in a conceptualization of motivation in terms of expectancies of danger and safety. It originally postulated two distinct learning processes, a punishment mechanism for rapid suppression of inappropriate SSDRs and a safety-signal reinforcement process governing the gradual acquisition of non-SSDRs. Both were discarded (1975) in favour of the view that SSDRs are respondent behaviours differentially elicited by supporting stimuli in the environment and that the gradual learning of non-SSDRs is governed primarily by motivational rather than reinforcing properties of SSs (safety-signals). (pp.207-208).

Two recent accounts of avoidance learning have included some aspects of the SSDR formulation in their analysis. These are the perceptual-defensive-recuperative model (Bolles & Fanselow, 1980) and the defense motivation system account

(Masterson & Crawford, 1982).

The class of behaviours termed SSDRs has been extended with the finding that rats' administered a single electric shock through a prod reliably bury the prod rather than flee, fight or freeze (Pinel & Treit, 1978; Blampied & Kirk, 1983). This class of behaviour has been termed conditioned defensive burying (for review see Pinel & Treit, 1982).

While it can be argued that Bolles has achieved what he set out to do, i.e., adequately explain the response problem in avoidance learning, SSDR theory of avoidance learning is not without its limitations and criticisms.

First, let us examine the response problem: is it a problem as Bolles has stated? The SSDR analysis places constraints on what behaviours will be readily acquired as conditioned avoidance responses with Bolles highlighting the difficulty in achieving rapid lever-press avoidance acquisition as empirical evidence in support of SSDR theory. However, there is now considerable evidence that lever-pressing can be acquired reasonably rapidly as a conditioned avoidance response under certain circumstances, thus placing lever pressing at an intermediate point on the continuum of difficulty. If rats can escape from the grid-floor to a 'safe' place or spend the ITI outside the conditioning chamber then lever-pressing is facilitated in comparison to control animals (Masterson, 1970; Crawford & Masterson, 1978). Other circumstances which facilitate lever-pressing avoidance acquisition include precedures which shorten the ITI (Pearl, 1963; Pearl & Fitzgerald, 1966; Cole & Fantino, 1966), use discontinuous shock (D'Amato, Keller & Di Cara, 1964; Berger & Brush, 1975); use of a retractable lever, (i.e.,

the lever is withdrawn from the experimental chamber during the ITI), (Cole & Fantino, 1966; Christopherson & Denny, 1967) which has the effect of preventing lever-holding during the ITI (Davis & Burton, 1974, 1976; Peterson & Lyon, 1975); use of non-contingent shock (Feldman & Bremmer, 1963; Delprato & Holmes, 1977); increasing the signal-shock interval (Berger, 1969; Berger & Brush, 1975); response shaping of the lever-press avoidance response (Keehn & Webster, 1968; Guilian & Schmaltz, 1973); punishment of freezing or lever-holding (Feldman & Bremmer, 1963); and lever-press-contingent grid floor termination (Black, 1980).

A much reported finding is that one-way avoidance acquisition is learned more rapidly than two-way avoidance acquisition (Moyer & Korn, 1966; Theios & Dunaway, 1964; Theios, Lynch & Lowe, 1966). Again, research has shown that under circumstances, e.g., when two-way avoidance responses produce grid-floor termination, they are learned as rapidly as one-way avoidance responses (Boyd & Levis, 1979; Black, 1980; Kostanek & Sawrey, 1965; Modaresi, 1975, 1978).

To explain facilitated two-way avoidance performance, Modaresi (1978) took the eclectic theoretical approach, as he explains:

> Thus, availability of the platform instead of the grid floor following a response is expected to produce a greater reduction in fear (McAllister, McAllister, & Dieter, 1976), a greater magnitude of stimulus change between start and goal compartments (Bolles & Grossen, 1970), a more effective inhibition of fear (Weisman & Litner, 1971), a more discriminable cue informing subjects that an avoidance response has successfully been made (D'Amato, 1970), or a facilitated relaxation

> response (Denny, 1971). Lumping all these hypothetical mechanisms together, the view suggesting a facilitating effect due to presence of the platform after criterion responding will, hereafter, be referred to as the reinforcement hypothesis. (pp. 84-85).

While Modaresi (1978) is probably correct in his theoretical analysis, the important point made by the research just reviewed is that Bolles (1970, 1972) has overstated the response problem in avoidance learning, and given particular circumstances non-SSDRs, e.g., lever-presses and two-way shuttle responses, can become conditioned avoidance responses.

Masterson and Crawford (1982) and Crawford and Masterson (1982) have criticized SSDR theory for its lack of a reinforcement principle. The Crawford and Masterson (1978) data clearly showed that performance of a SSDR, per se, e.g., flight, was not necessary for rapid acquisition of lever-pressing, but rather the stimulus changes associated with removal from an aversiveness environment to a safe place proved crucial for avoidance acquisition and maintenance.

Finally, a problem with SSDR analysis is its inflexibility as to what constitutes an SSDR, giving it a post hoc character. As Fantino and Logan (1979) note:

> It is too easy, after the fact, to make intuitive guesses as to what is a "natural" or a "highly probable" response and what is not. What is needed is careful parametric work in which the probabilities of various responses to a given UCS are ascertained prior to studying the efficacy of these responses in avoidance. (p. 266).

Preparedness Theory

Bolles' SSDR theory linked psychological and ethological approaches to animal learning. Since then other theorists have developed ethologically relevant theories (Bolles & Fanselow, 1980; Masterson & Crawford, 1982; see Johnston, 1981 for review), including preparedness theory (Seligman, 1971). Such theories fall into the "biological boundaries of learning" approach which began with the conditioned taste-aversion literature (Garcia & Koelling, 1966). This theoretical line of explanation developed in part from criticisms of the conditioning model of avoidance, fear and phobia acquisition (Seligman, 1971; Eysenck, 1976; Rachman, 1978; Bandura, 1977).

McNally and Reiss (1982) succinctly summarized preparedness theory as follows:

> First phobias are assumed to result from Pavlovian conditioning so that initially neutral stimuli (CSs) become fear-eliciting when experienced in temporal contiguity with aversive events (USs). Second, CSs are graded along a continuum of preparedness, or biological predispositions, for fear conditioning. This implies that some CSs are much more likely than others to become phobic. Third, preparedness is viewed as a result of evolution and natural selection so that the CSs assumed to be highly prepared for fear conditioning are those which were dangerous to pre-technological people. (p. 53).

Seligman (1971) and others have argued that empirical support for preparedness theory has come from the following research areas:

1. The inability of English (1929) and Bregman (1934) to replicate Watson and Rayner's (1920) classical fear

conditioning study. A number of theorists (Eysenck, 1976; Marks, 1969; Rachman, 1977; Seligman, 1971) have argued that English and Bregman used evolutionary neutral, non prepared, CSs, e.g., household objects and wooden blocks, which are stimuli not genetically predisposed to become fear evoking whereas Watson and Rayner used a rat CS which being a prepared stimulus lead to the rapid acquisition of a fear reaction. Delprato (1980) argues it is incorrect to use the results of English (1929) and Bregman (1934) as supportive evidence for preparedness theory because of methodological weakeness in these studies. English (1929) failed to show the UCS as being functionally effective, therefore it is not surprising that the CSs failed to elicit a conditioned fear reaction. Bregman (1934) paired neutral CSs with either a loud bell (aversive UCS) or a rattle, melody (positive UCS) using a within subject design. Bregman failed to independently evaluate if the bell was aversive or the rattle and melody positive, therefore it is difficult to ascertain if this distinction really existed. Bregman also failed to obtain differential responding to the CSs. Given these problems with the above studies it is unwise to use the English and Bregman results to refute Watson and Rayner's findings.

2. The research associated with the conditioned taste-aversion literature has been cited as supporting preparedness theory (Seligman, 1970, 1971; Seligman & Hager, 1972). Delprato (1980) summarizes the importance of this literature to preparedness theory as follows:

> The major implication of the taste-aversion paradigm is that contrary to assumptions of conventional learning views, due to natural selection, different preaversive stimuli are

> not equally likely to come to evoke fear when they signal a given aversive event ... The phenomena revolving around taste-aversion behaviour cannot be ignored, denied or viewed as anomalous (Rozin, 1977), but critical examination reveals that neither do they unequivocally support the notion that genetically inherited behavioural predispositions are involved in the establishment of taste-aversions. (pp.84-85).

The adaptive-evolutionary interpretation of taste-aversion learning adopted by adherents of preparedness theory has been criticized by some on methodological grounds (Bitterman, 1976; Spiker, 1977; Testa & Ternes, 1977) but defended by others (Garcia, Hankins & Rusiniak, 1976; Revusky, 1977) with the situation being left unresolved.

The taste-aversion paradigm (Milgram, Krames & Alloway, 1977; Barker, Best & Domjan, 1977) has also been used by supporters of preparedness theory in their criticism of the equipotentiality principle of conditioning. The concept of equipotentiality implies that one CS should act as any other in fear conditioning as well as one response like any other response should be acquired as a conditioned avoidance response. Levis (1979) points out that the concept of equipotentiality holds true only when everything else is held equal. While preparedness theory through its adaptive-evolutionary position can explain why some stimuli such as spiders and snakes are acquired more readily as fear evoking than others such as shoes or flowers, the CS pre-exposure effect, also called latent inhibition (Lubow & Moore, 1959) from traditional conditioning theory can also account equally well for the CS effect.

3. Bolles' SSDR analysis identifying response topography

as being a critical factor in avoidance acquisition has been used as evidence against the equipotentiality principle and in support of preparedness theory (Seligman & Hager, 1972). As has been previously discussed the difficulty encountered in acquiring lever-press avoidance may be a function of the experimental contingencies in operation rather than the status of the lever-pressing non-SSDR or the evolutionary non-significance of bar-pressing. In a summary of the response topography research in support of the adaptive-evolutionary approach to avoidance learning, Delprato (1980) states:

> A major characteristic of the emphasis on hereditary factors is the hypothesis that the topography of avoidance responses must be compatible with innate defensive behaviour if avoidance is to be learned ... a number of findings have been viewed as consistent with this hypothesis. However, several considerations make it difficult to unreservedly recommend the hypothesis at this time. Problems of circular responding, failure to control subjects' developmental histories, efficient lever-press avoidance produced in recent experiments, failure to consider training conditions, and confounding of environmental variables with response topography suggest limitations on the adaptive-evolutionary view of avoidance learning. (p. 93).

While preparedness theory can explain data which offers interpretive difficulties for conditioning theory, more experimental research is required with consideration given to the methodological criticisms raised above.

## Cognitive theories of avoidance

Tolman (1932, 1934) integrated the empirical results from classical conditioning, instrumental trial-and-error

learning and the higher learning processes into one theoretical system. Tolman developed his cognitive theory of learning at a time when the behaviourist viewpoint was dominant in American Psychology. While Tolman's analysis was not specifically developed to account for avoidance learning, Solomon and Brush (1956) have applied his cognitive theory to avoidance learning as follows:

> ... his point was that conditioning and learning were characterized by perceptual reorganization by changed associations among sensory events. He felt that the CS-US relationship in Pavlovian conditioning established in S expectations as well as knowledge, about what is followed by what. The CR is an index of the strength of the expectation that the US will always follow the CS. The CS comes to symbolize or forecast the US, and so it achieves some of the functional properties of the US... Anxiety for Tolman is not a drive state but a negative expectation, a knowledge of bad things to come. (pp.236-237).

Tolman's cognitive theory had little impact on Psychology, being formulated against the backdrop behaviourist tradition. Osgood (1950) put forward a number of theoretical criticisms to refute Tolman's theoretical analysis of avoidance learning. However, with the advent of a cognitive science approach to human information processing (Anderson, 1976), cognitive-expectancy interpretations of learning processes in animals have begun to flourish (Premack & Woodruff,1978; Hulse, Fowler & Honig, 1978; Roitblat, 1982; Dennett, 1983; Roitblat, Bever & Terrace, 1983). This is also true of avoidance learning with a number of cognitive-expectancy theories having been proposed.

Seligman and Johnston's Cognitive-Expectancy Theory

This theory based on Irwin's (1971) cognitive theory of motivation ascribes the role of conditioned fear to the CS only in the early phase of avoidance acquisition, at asymptotic avoidance responding, conditioned fear no longer plays a motivating role and is replaced by two expectancies that maintain responding, firstly, that avoidance responding produces no aversive consequence, and secondly, that not responding produces aversive consequences. Integration of these expectancies produces a preference for responding over not responding. Seligman and Johnston (1973) define expectancy and preference as follows:

> ... an expectancy is a hypothetical construct: a state of the organism which represents (stores information about) contingencies between responses and outcomes in a given situation ... A preference is also a hypothetical construct: a state of the organism which controls the choice of response on the basis of outcomes expected. (p. 90).

This theory predicts the low correlation between autonomic nervous system arousal and avoidance performance found in the literature (Black, 1959; Solomon & Wynne, 1954; Bersh, Notterman & Schoenfeld, 1956; Werboff, Duane & Cohen, 1964).

In limiting the motivating role of conditioned fear to early avoidance acquisition, Seligman and Johnston's theory cannot explain accelerated avoidance performance due to fear-excitatory CSs, if such performance is independent of conditioned fear (Rescorla & LoLordo, 1965). Masterson and Crawford's (1982) criticisms of expectancy-type analyses have

already been noted above (pp. 23-24).

Bolles (1972b, 1978) has also proposed an expectancy analysis of avoidance behaviour. Bolles proposes all learning involves the acquisition of knowledge, expectancies, about S-S* and R-S* contingencies where S = Stimulus, R = response, S* = biological significant event and the linkage between S-S* and R-S* being a temporal parameter. In avoidance responding, the S* of the S-S* expectancy is an aversive event, and the S* of the R-S* expectancy is absence of the aversive event, i.e., safety. Thus the stimulus, in absence of responding produces an aversive event, while responding leads to safety. In Bolles expectancy analysis the S-S* expectancy performes a motivational role, somewhat similar to conditioned fear in two-process learning theory, while in Seligman and Johnston's cognitive theory there are no motivational components. In his expectancy analysis Bolles has termed SSDRs as the behavioural response patterns of underlining innate R-S* (Safety) expectancies. Thus, performing a functionally effeetive SSDR avoidance response (freezing, fleeing, fighting) leads to confirmation of R - safety expectancies. If SSDRs are ineffective as avoidance responses then the R - safety expectancy is disconfirmed and weakened being replaced by a R - aversiveness expectancy, i.e., responding produces shock not safety. Bolles proposes that if innate R - safety expectancies are disconfirmed then it is difficult for the subject to learn non - SSDR R - safety expectancies, and that this explains why some avoidance responses are more difficult to acquire than others. However, this position seems counterintuitive from an adaptive-evolutionary viewpoint and is also not supported by the

evidence that non SSDRs can be acquired as avoidance responses.

Reiss (1980) has recently developed an expectancy model of Pavlovian conditioning which he has applied to the acquisition of human fear-phobic reactions. He outlines his model as follows:

> The model maintains that what is learned in Pavlovian conditioning is an expectation regarding the occurrence, or nonoccurrence, of a US onset or a change in US magnitude or duration. Expectancies are considered to be mediating responses with covert stimulus properties that can become elicitors of a number of anticipatory responses. These behaviours include the CR, verbal reports of CS-US relations ("awareness"), and instrumental responses including approach and avoidance ... A four-process model is proposed consisting of danger expectancies, anxiety expectancies, negative reinforcement of avoidance behaviour and self-reinforcement of avoidance behaviour. (pp.387-388).

Danger expectancies are viewed as expectancies anticipating physical or social danger and result from cognitive and associative learning, covert conditioning, observations of models or any combinations of the above. An anxiety expectancy is an expectancy of anxiety when in the presence of certain stimuli. Negative reinforcement of avoidance is by the process of anxiety reduction, which is also proposed in two-process theory and its modifications. Self-reinforcement of avoidance, according to Reiss, is achieved primarily by feedback stimuli acting as conditioned inhibitors of fear and/or safety-signals. Thus, this aspect of Reiss' expectancy model is equivalent to the conditioned inhibition and safety-signal modifications of two-process theory.

Recently, Burgess (1981) has critiqued Reiss' expectancy

model and outlined several inaccuracies of his model which lead Burgess to reject Reiss' expectancy model. Burgess outlined two problems which all expectancy analyses have in common, firstly,

> ... its (expectancy notion) use is usually circular. That is, it is defined by the very-behaviour it is purported to explain (Levis, 1976; Osgood, 1953)... Reiss' specification of the conditions under which expectancies are formed is vague at best ... Several questions may be posed. For example, what happens if there is a CR but no approach or avoidance behaviour emerges? ... Does such a result indicate the presence of absence of an expectancy? How strong must an expectancy be for it to influence behaviour? What kind of temporal gaps can expectancies bridge (i.e., how important is temporal contiguity)? (p. 11).

and the second problem associated with an expectancy analysis,

> ... is that it tends to lead to a circumvention of an experimental analysis of behaviour. It achieves this effect for two reasons. First, it gives the impression that it has provided a full causal account of the behaviour when in fact it has not, and second, the variables which control the behaviour are left untouched because they are supposed to reside inside the skin of the organism (Henton and Iversen, 1978; Skinner, 1966). (p. 11).

Summary

A large number of theoretical accounts of avoidance learning have been reveiwed. It is clear that no one theory accounts for all the empirical research on avoidance behaviour. This is true even of Modaresi's (1978) account which combined five different theories into a single reinforcement theory of avoidance behaviour. The theories have been either

contiguity-conditioning based or expectancy-cognitive based. This dichotomy lead Wolpe (1981) to propose that fear motivated human neurotic behaviours develop either by direct autonomic conditioning or the establishment of new cognitive associations to already present anxiety motivated phobic behaviours. Clearly, as Mackintosh(1974) states, the issue becomes:

> ... one of whether avoidance behaviour is mediated by classically conditioned motivational states, or by the development of expectations about the consequences of responding. (p. 330-331).

One final point, while it is common to find the procedural distinction made between Pavlovian and Operant conditioning methods, it is important to remember that in all operant conditioning procedures there are Pavlovian conditioning contingencies and they are more obvious in the avoidance/escape paradigm.

Theory and method in avoidance learning: Extinction processes

Extinction of avoidance responses is typically operationalized by removing the aversive stimulus while continuing to present the conditioned stimulus. Normally the subject continues to escape the CS during the early phase of extinction as there is no opportunity to discriminate between acquisition and extinction procedures. Mowrer's two-process theory maintains that conditioned fear does not change because fear is generated by CS presentations and reduced by avoidance responding, resulting in reinforcement and persistence of the avoidance response. By removing itself from prolonged CS exposure, the subject removes itself from the situation in which new learning of the new CS-UCS contingency might take place, and Mowrer (1950) has referred to this behaviour as the "neurotic paradox" relating it to human neurotic behaviour which is often self-defeating and self-perpetuating. However, persistence of responding in extinction, as we have previously noted, presents a problem for two-factor theory because over a series of consecutive avoidance responses, the fear CR should gradually extinguish because each extinction trial, in essence, is a Pavlovian extinction trial where the CS is presented in the absence of the UCS. As previously noted, Solomon and Wynne (1954) proposed the principles of anxiety conservation and partial irreversibility of the CR to explain the resistence-to-extinction findings. The anxiety conservation principle was later modified by Stampfl (1960) incorporating the proposition of serially-ordered CSs increasing CS complexity and resistance-to-extinction. According to Stampfl's modified

version of two-process theory, fear extinction to CSs temporally most distal from the UCS takes place first, followed by extinction to CSs closer temporally to the UCS. Finally, the CS complex will fail to elicit conditioned fear thus producing extinction of avoidance responding.

Fear extinction to the CS complex is a necessary and sufficient condition for two-process theory to produce avoidance extinction but for effective reinforcement theory, both fear extinction to the CS and situational cues must occur for the avoidance response to extinguish.

Expectancy-cognitive models of avoidance extinction require the expectancy that non-responding leads to presentation of aversive stimuli be modified to the expectancy that absence of responding will not be followed by an aversive consequence. In the following section, I examine these theories of extinction as applied to research methods designed to facilitate the hastening of avoidance extinction, an issue which has clear relevance to clinical practice.

## Procedure and theory in facilitation of avoidance extinction

Because of similarities between experimentally produced avoidance behaviour in animals and fear motivated human neurotic behaviours (Baum & Poser, 1971; Stampfl & Levis, 1967; Leitenberg, 1976) as well as the empirical evidence that both animal and human avoidance behaviour is highly resistant to extinction processes (Baum, 1965; Levis, 1970, 1974) research and clinical attention has focused on investigation of techniques for eliminating avoidance behaviour and reducing fear which accompanies such behaviour.

These techniques have included conventional extinction which is simply a continuation of acquisition trials except the UCS is never presented (Solomon, Kamin & Wynne, 1953); presenting the UCS during extinction when the subject makes an avoidance response (Baum, 1970b), the non-termination of the CS after the avoidance response (Katzev, 1967); response prevention whereby the avoidance response is prevented while simultaneously exposing the subject to the CS over an extended time interval (Baum, 1969a,1970b; Weinberger, 1965; see Kirk & Blampied, 1980, for a bibliography of response prevention research); and counterconditioning based on Wolpe's (1958) reciprocal inhibition principle. In this procedure a response antagonistic to anxiety (e.g., eating, relaxation) is paired with exposure to fear provoking stimuli in a graded manner from least to most aversive (Wolpe, 1958, 1982). Most research has focused on response prevention or flooding and counterconditioning techniques for facilitating extinction of avoidance behaviour.

The response prevention technique has been used with human clinical populations under the names of Implosive therapy, flooding and response prevention (Baum & Poser, 1971; Hogan, 1969; Morganstern, 1973; Smith, Dickson & Sheppard, 1973; Stampfl & Levis, 1967; Marshall, Gauthier & Gordon, 1979; Adams & Hughes, 1976; Levis & Hare, 1977). The terms response prevention or flooding refer to a number of procedural techniques. Baum (1976) refers to the techniques as flooding - 1, flooding - 2 and flooding - 3 and describes them as follows:

> One method of administering flooding involves allowing the avoidance response to be made at will

> by the animal, but seeing to it that the CS is not terminated (e.g., Bankart, 1971; Heath, 1968; Polin, 1959; Shearman, 1970) ... a one-way avoidance situation (see Baum, 1966), flooding - 1 involves beginning a new trial as soon as the response is made; thus it entails reducing the intertrial interval to zero ... flooding - 2, involves blocking or thwarthing the avoidance response while exposing the organism to the feared CS or cues ... flooding - 3 involves removing a part of the avoidance apparatus and in this way making it impossible for the animal to perform the avoidance response. (pp. 114-115).

In the present study, flooding - 1 is referred to as flooding (after Polin, 1959) while flooding - 2 and flooding - 3 are collectively referred to as response prevention (after Page & Hall, 1953).

Adams and Hughes (1976) differentiate procedural variations of response prevention as follows:

> In <u>response prevention</u> (RP), the CAR (conditioned avoidance response) is not allowed ... The <u>response prevention delay</u> (RPD) procedure utilizes similar methods, but the CAR is allowed to occur at some specified period after CS onset. In other words, response prevention occurs for a relatively brief period at the onset of each trial. Punishment response prevention (PRP) is another special type of response prevention. Prevention of CARs is achieved by making onset of an aversive stimulus contingent upon the occurrence of a CAR rather than by employing physical suppression. (p. 215).

For the purposes of the present review, attention is focused primarily on the procedures called flooding - 2 and flooding - 3 by Baum (1976) and on the response prevention procedure of Adams and Hughes (1976). Research on the

response prevention technique fits into three main categories, firstly parametric and procedural studies on the response prevention procedure itself, secondly, comparisons of response prevention with regular extinction, flooding or no-treatment controls, and thirdly, isolating factors responsible for facilitating the action of response prevention. I shall briefly review these research areas before discussing fear assessment procedures and theoretical analyses of response prevention.

## Parametric and procedural variants of the response prevention procedure

### Unconditioned stimulus intensity

Baum (1969a) directly mainpulated UCS intensity during avoidance acquisition and found increasing UCS intensity (0.5mA to 2.0mA) decreased the efficacy of response prevention and also decreased the amount of general activity and grooming behaviour exhibited during response prevention. While Baum (1969a) used the traditional method of reduction in the CAR, jump-up responding, as an index of fear reduction during extinction, Corriveau (1977) used a safety testing behavioural approach procedure, which he describes as follows:

> In the one-way platform apparatus ... avoidance trained subjects, when placed on the platform, would typically reach down from the platform and touch the grids several times before completely departing the platform. A typical fearful rat would hold itself on the platform with its hindpaws, reach down with it's front paws, touch the grids, rear completely back to the platform, and reach

down a few more times before cautiously approaching the grids. (pp. 1-2).

Using the safety testing approach procedure the dependant variables of interest are, the time taken to first engage in a safety test response, the frequency of safety testing responses before completely departing the platform and total platform time. Corriveau (1977) reported the group trained with the less intense shock approached the grids significantly sooner, spent significantly less total time on the platform and required significantly fewer safety test responses before approaching the grids in comparison to the other groups.

Tortora and Denny (1973) manipulated both UCS intensity (0.3, 0.8, and 1.8mA) and RP treatment duration (15, 60, 120 and 165 seconds). They report the effect of UCS intensity, except for 15- second RP, was the same as reported by Baum (1969a). Thus, Corriveau (1977) and Tortora and Denny's (1973) findings corroborate Baum's (1969a) study.

Response prevention duration

A number of investigators have shown increasing response prevention duration leads to hastening extinction of avoidance and fear behaviour (Baum 1969a, b; Bersh & Keltz, 1971; Coulter, Riccio & Page, 1969; Franchina, Agee & Hauser, 1974; Mineka, Miller, Gino & Giencke, 1981; Neill, 1983; Rohrbaugh & Riccio, 1970; Schiff, Smith & Prochaska, 1972; Tortora & Denny, 1973; Weinberger, 1965). Determining what duration of response prevention treatment is necessary and sufficient to reduce avoidance and fear behaviour is extremely difficult and complex as the following passage from Mineka et al., (1981) illustrates:

> In using a prolonged period of flooding (60 min), Corriveau and Smith (1978) were apparently interested in demonstrating that flooding can reduce fear, and by including nonavoidance trained control groups they could assess whether prolonged flooding produced complete fear reduction ... However, an interesting question left unanswered by their experiment, is whether smaller amounts of flooding sufficient to hasten jump-up avoidance response extinction would also be sufficient to reduce fear as assessed by their techniques. The results of Mineka and Gino (1979) using a two-way shuttlebox avoidance response and the CER as an index of fear suggest the possibility that small amounts of flooding may have significant effects on avoidance response extinction but not on other indices of fear. Alternatively, as suggested by Baum (1970) and Mineka (1979), it is possible that different mechanisms are responsible for mediating the effects of flooding different kinds of avoidance responses and so a different, or even opposite, pattern of results might emerge for the jump-up box. p.(437).

Immediate vs delayed response prevention

The rationale for investigating immediate versus delayed administration of response prevention following conditioned avoidance acquisition has been if delayed RP is less effective in reducing fear than immediate administration then the implication for implosive and flooding therapies based on RP procedures would be obvious. Intuitively, immediate treatment of fear motivated phobic behaviours seems the most efficient procedure to eliminate conditioned fear. Delays in treatment could produce incubation of fear (Eysenck, 1968, 1979) requiring more prolonged and complex treatment. Yet, Baum (1976) argued that RP could be more effective if delayed

because the subject would be in the preferred quiescent state at the beginning of RP rather than being agitated. Baum (1972a) reported a 5 minute RP session hastened extinction of avoidance responding more effectively when administered 30 minutes after avoidance acquisition rather than immediately after. Neill (1983) administered RP at delays of 1 minute, 1, 7, and 49 days. He reported no significant treatment delay interval effect, that is, a delay of 49 days was just as effective at eliminating avoidance responding as a delay of 1 minute.

Iso (1979) delayed distributed - RP administration by either 0, 4, or 24 hours following avoidance training. Response prevention was relatively ineffective in eliminating avoidance behaviour at a delay of 4 hours, but effective in hastening extinction with delays of 0 or 24 hours. Gordon, Smith and Katz (1979) administered a brief 15-second RP treatment either 24, 72 or 96 hours after active avoidance training in one of a series of experiments. Response prevention at 24 hours had no effect on avoidance latencies in comparison to controls while RP at 72 and 96 hours following avoidance training resulted in enhanced rather than decreased performance of avoidance responding. In another experiment, Gordon et al. (1979) reported RP given 0.25 hours following avoidance acquisition significantly decreased avoidance latencies in comparison to a control group, while RP at 72 hours resulted in enhanced avoidance performance. It is clear from the above literature that further research is required to delineate the processes in operation which result in different findings when the period between avoidance acquisition and response prevention treatment is given.

Repeated Administration of Response Prevention

Baum (1972b) repeated the normal experimental sequence of avoidance acquisition - response prevention - extinction daily for five days. He reported RP was effective in hastening extinction of avoidance behaviour on days 1, 2 and 3, but had no effect on days 4 and 5. Akiyama (1968, 1969) also found diminished efficacy of RP on the second repetition of the acquisition - RP - extinction sequence.

Massed versus Distributed Response Prevention

Polin (1959) reported massed RP to be superior to distributed RP in hastening avoidance behaviour. Shearman (1970) believed Polin's results were clouded by methodological weaknesses in his experimental design and replicated his study with a more vigorous design. Shearman (1970) reported no significant differences between massed vs distributed RP, a result subsequently confirmed by Bankart and Elliott (1974). Schiff, Smith and Prochaska (1972) varied the number and duration of RP trials and reported that total RP time, as opposed to massed vs distributed RP, was the critical factor for effective hastening of extinction by the RP procedure. In contrast Baum and Myran (1971), Berman and Katzev (1972) and Franchina, Agee and Hauser (1974) all reported superiority of distributed RP over massed RP in hastening extinction of avoidance performance. These discrepancies between the studies in part reflect procedural differences between them (for discussion see Monti and Smith, 1976). Monti and Smith (1976) explain the superiority of distributed RP over massed RP in terms of Rescorla and Solomon's (1967) contingency

theory,

> which holds that extinction of conditioned fear is a function of CS-US contingency reduction. Since each nonreinforced presentation of the CS reduces the CS-US contingency, this theory would appear to predict that extinction of conditioned fear is largely a function of the number of nonreinforced CS exposures with exposure duration having little effect. (p. 150).

Yet, other studies have found non-reinforced CS exposure duration to be critical (Mineka & Gino, 1979; Schiff, Smith & Prochaska, 1972; Shipley, 1974). In her review of RP studies, Mineka (1979) summed up the role of non-reinforced CS exposure duration as follows:

> ... These results all suggest that total non-reinforced CS exposure - presumably allowing for fear extinction to occur - may be the crucial variable in producing rapid extinction of avoidance. It must be emphasized, however, that this conclusion is based on the as yet unsupported assumption that the amount of fear extinction of an avoidance CS is directly related to the amount of non-reinforced CS exposure ... as yet no one has studied this issue directly in the avoidance/flooding situation, i.e., whether fear extinction of an avoidance CS (as measured by the CER test) is a simple function of total amount of non reinforced CS exposure. (p. 996).

Overtraining Avoidance Acquisition and Response Prevention

Baum (1968) varied the number of overtraining trials (0, 50 or 100) subjects received after achieving an avoidance acquisition criterion, but before RP administration. Baum reported overtraining (50 or 100) increased persistence of responding during extinction only if subjects failed to avoid

during the extra training trials. Other investigators also report overtraining leads to more resistance to extinction with a jump-up avoidance response task (Buss & Reid, 1973; Cooper, Coon, Mejta & Reid, 1974; Mineka, 1978; Sautter & Reid, 1973; Voss, Mejta & Reid, 1974). Buss and Reid (1973) and Voss, Mejta and Reid (1974) reported positive hypothalamic intracranial stimulation (ICS) during response prevention mitigated the overtraining effect, i.e., subjects receiving ICS and RP responded reliably less during extinction than subjects that received RP alone. Mineka (1978) further assessed the effect of overtraining in a shuttlebox avoidance response procedure. Overtraining failed to reduce the efficacy of RP using a shuttlebox avoidance procedure. Clearly, the type of avoidance response task selected determines if overtraining avoidance responding will later effect avoidance extinction.

UCS Presentations During Response Prevention

Normally, RP involves exposing subjects to the CS-complex in the absence of the UCS for a specific period of time. A number of studies have investigated the effects of UCS presentations during RP (Marrazo, Riccio & Riley, 1974; Bersh & Miller, 1975; Bersh, Whitehouse & Mauro, 1982). Marrazo et al. (1974) reported RP was just as effective in hastening extinction with additional inescapable UCS presentations (Pavlovian fear conditioning) as without them. Bersh and Miller (1975) replicated Marrazo et al. (1974) and found their result was dependent on UCS duration (5 seconds) employed during RP. Bersh and Miller (1975) used both 0.5 and 5.0 second UCS durations and found brief US presentations

(0.5 sec.) during RP enhanced resistance to extinction of avoidance responding in comparison to RP alone subjects. Bersh et al. (1982) further investigated the effect of Pavlovian fear conditioning during RP upon subsequent avoidance response extinction. In addition to no-RP control, RP-alone, RP + UCS groups, Bersh et al. (1982) included a group which received RP + UCS presentations in the presence of a light - CS plus the opportunity to escape both CS and UCS on the final RP trial. Bersh et al. reported no reliable difference between control and RP + UCS subjects, while RP-alone subjects exhibited facilitated avoidance extinction. For subjects given CS - US pairing during RP, opportunity to escape the CS - US complex increased subsequent resistant to extinction in comparison to all other groups, while absence of the opportunity to escape lead to rapid avoidance extinction. Further experimentation confirmed the above results in part reflecting stimulus generalization from both acquisition and response-prevention phases to the extinction phase of the experiment (Bersh et al., 1982, experiment 2).

Avoidance Re-acquisition Interpolated Between Response Prevention and Extinction

Bersh and Keltz (1971) and Franchina, Agee and Hauser (1974) reported that following a single avoidance reinstatement trial, response prevention facilitated avoidance extinction, but not as much as control subjects which received RP-alone before extinction testing. Franchina, Hauser and Agee (1975) administered 10 avoidance reinstatement trials prior to extinction and reported that reinstatement training decreased the facilitating effect of RP on avoidance extinction.

In a slightly different procedure to that of the above studies, Franchina and Meyers (1976) interpolated avoidance-escape trials between distributed RP trials. Franchina and Meyers (1976) reported attenuated facilitation of avoidance extinction when distributed-RP consisted of interpolated avoidance-escape trials.

Fear Enhancement Following Brief Response-prevention

Brief RP exposures following avoidance acquisition have been shown to enhance avoidance responding in extinction (Prado-Alcala, Bush, Steele & Reid, 1978; Gordon, Smith & Katz, 1979), to increase response suppression in a CER procedure (Rohrbaugh, Riccio & Arthur, 1972) and exacerbate fear of CS cues when using approach measures, passive avoidance, to assess fear (Linton, Riccio, Rohrbaugh & Page, 1970; Rohrbaugh & Riccio, 1970). Reid, Taylor and Rassel (1971) reported that brief aversive ICS during RP enhanced or sensitized persisting avoidance responding during extinction while Prado-Alcala et al. (1973) found this enhancement effect could be eliminated by ICS delivered contingently upon moving away from the ledge of a jump-up apparatus during RP. This enhancement of fear-related responses has been incorporated into an incubation theory of avoidance and neurosis (Eysenck, 1968, 1979) which is discussed in detail later. Neill (1983) reported brief RP durations failed to produce fear enhancement, measured using the approach assessment methodology (Corriveau & Smith, 1978; Bersh & Paytner, 1972; Mineka et al., 1981), so is in disagreement with the above studies.

Neill (1983) while finding no overall fear enhancement effect, did report brief RP durations did produce greater

fear in approach to the grid floor of a jump-up apparatus than no treatment control at a 1 minute RP treatment delay interval, but not at intervals of 1, 7 or 49 days. This finding is in agreement with Rohrbaugh et al., (1972) but in conflict with the no enhancement effect with immediate RP following avoidance acquisition reported by Rohrbaugh and Riccio, (1970). Neill (1983) discussed the results in terms of a Kamin effect (Kamin, 1957; Brush, 1971; Pinel & Cooper, 1966; Spear, 1973) and the parameters involved, e.g.,

> ... A more plausible explanation is that the parameters employed to induce increased fear in this experiment were insufficient to do so. While both Gordon et al., (1979) and Rohrbaugh, et al., (1972) were able to demonstrate enhancement after a 15 sec. CS exposure, a series of experiments by Rohrbaugh & Riccio (1970) suggest that the enhancement effect can be somewhat elusive. In their first study no enhancement of fear was demonstrated after a 30 sec. CS exposure while 5 min. of RP increased fear only marginally. However, by employing younger subjects and a weaker shock level ... they were able to show a robust enhancement of fear after both 30 and 60 sec. of RP and no enhancement at five min. of CS exposure. (p. 42).

The Kamin effect was discussed by Kamin (1957) who reported that subjects trained to actively avoid a CS showed performance decrements if training was interrupted and resumed 1 hour later. These decrements were not present if the interruption lasted for 24 hours.

Response Prevention and Residual Fear

In a number of studies, subjects received RP treatment

subsequently exhibited residual fear to CS-cues when approach assessment procedures were used (Coulter, Riccio & Page, 1959; Dickson, Sisemore, Andert, Hustak & Quillin, 1977; Linton, Riccio, Rohrbaugh & Page, 1970; Page, 1955; see Riccio & Silverstri, 1973 for a review). Page (1955), and Coulter, Riccio and Page (1969) found RP treated subjects took reliably longer to return to the CS complex than regularly extinguished subjects. Linton, et al., (1970) eliminated the extinction phase from their experiment and tested for residual fear following RP or no RP treatment using a passive avoidance test. Again the RP treated animal took reliably longer than controls to return to the CS-complex. Baum (1971) following avoidance acquisition, RP or no-RP, and avoidance extinction, presented a loud buzzer to all subjects. The loud Buzzer induced responding to recover from extinction (disinhibition) in both RP and no-RP treated subjects, indicating residual fear was present across groups. Taken together, the above studies indicate the absence of avoidance responding as measured by extinction criteria does not reliably reflect the underlying motivational state of subjects, a conclusion supported by Baum (1971):

> While it is easy to control avoidance behaviour through response prevention or even by means of regular extinction procedures, underlying fear which is established in the process of avoidance acquisition is much more difficult to manage or eliminate. (p. 208).

Reynierse, Klomp and Bach (1974) investigated the effects of RP on the "Kamin effect" in which relearning of the shuttlebox avoidance response took place either 0, 1, or 24 hours following RP administration. Response prevention

treatment reduced but did not eliminate the Kamin effect, indicating the presence of residual fear in RP treated subjects. Reynierse et al., (1974) proposed "Kamin effect" experiments as an alternative RP assessment procedures as follows:

> Methodologically, the Kamin effect is potentially important for assessing the effectiveness of response prevention procedures since it represents a preparation in which conditioned fear effects are relatively independent of the effective instrumental response. (p. 419).

## Techniques Facilitating the Efficacy of Response Prevention

While considerable research has shown that extended response prevention hastens avoidance extinction when fear is assessed by persistance of avoidance (Baum, 1968a, 1968b; Berman & Katzer, 1972; Mineka & Gino, 1979a) or assessed using the CER procedure (Monti & Smith, 1976; Mineka & Gino, 1979b) or assessed using approach assessment procedures (Bersh & Paytner, 1972; Corriveau & Smith, 1978; Corriveau, Contildes & Smith, 1978; Mineka, Miller, Gino & Giencke, 1981) a number of investigators have focused on techniques used in conjunction with RP that might enhance RP effects on subsequent avoidance extinction.

### Drugs as Adjuncts to Response Prevention

In general, research investigating tranquilizer-assisted RP has failed to show that tranquilizers (amobarbital, chlordiazepoxide, or chlorpromazine) increase the effectiveness of RP (Cooper et al., 1974; Christy & Reid, 1975; Kamano, 1968, 1972; Nelson, 1967). Taub et al., (1977) succinctly summarized the research as follows:

> Our initial testing of psychotropic agents as adjuncts to RP ... confirmed the results of others ... indicating that some drugs used as adjuncts to RP either reduced the usual effectiveness of RP or led to heightened persisting avoidance. Chlorpromizine, chlordiazepoxide, and sodium amytal at some doses, calmed the rat during RP but subsequently led to more persisting avoidance than would have been expected had the rat received no RP. (pp. 67-68).

The stimulant, amphetamine, has been reported to facilitate RP (Cooper et al., 1974; Christy & Reid, 1975), or have no effect (Taub, et al., 1977). Baum (1971) reported methylphenidate, a stimulant, had no effect on the efficacy of RP treatment. Taub et al., (1977) administered a number of drugs together with RP and found that atropine was the only drug that led to less persisting avoidance than RP controls. Following Taub et al., (1977) Blampied (1979) used the cholinergic blocking agent scopolamine but failed to find any effect on RP treatment.

In summary, the animal research literature has provided little support for the proposition that drugs, in particular, relaxing or anti-anxiety agents, maybe effective in facilitating RP in hastening avoidance-fear-anxiety extinction. The human research literature also contains some disappointing findings. The benzodiazepine tranquiliser diazepam has been reported to both facilitate progress in flooding sessions (Friedman & Lipsedge, 1971; Hussain, 1971; Johnston & Gath, 1973; Marks, Viswanathan, Lipsedge & Gardner, 1972; Razani, 1974) and have no effect (Whitehouse, Robinson, Blackwell & Stutz, 1978; Hafner & Marks, 1976). Other drugs which seemingly fail to enhance the effects of

in vivo flooding include mono-amine oxidase inhibitors (MAOI), (Solyom, Solyom, La Pierre, Pecknold & Morton, 1981), and beta-adrenergic blocking agents (Hafner & Milton, 1977). Experiments reported in this thesis further investigate the proposition that anti-anxiety agents maybe effective in facilitating RP.

Intracranial Stimulation (ICS)

While drug-assisted RP research showed little promise in increasing the efficacy of RP, the use of positive intracranial stimulation (ICS) of the posterior lateral hypothalamus has been reliably effective in facilitating RP enhanced avoidance extinction (Buss & Reid, 1973; Gordon & Baum, 1971; Hunsicher, Nelson & Reid, 1973; Baum, Leclerc & St Laurent, 1973; Metja, Reid, Coon, Paxton, Prado-Alcala, Christy, Ganey & Miller, 1974; Prado-Alcala, Brush, Steele & Reid, 1973; Reid, Miller, Stone, Monico, Rassel, Taylor & Sautter, 1973; Voss, Mejta & Reid, 1974). Metja et al., (1974) summarized the results of their experiments as follows:

> The accumulated data allows us to conclude with confidence that lateral hypothalamic medial forebrain bundle ICS when given during RP effectively counter-conditions persisting avoidance ... A positive affective stimulus must be presented only under certain circumstances for it to be an effective counter conditioner of persisting avoidance. (p. 31).

Metja et al., (1974) reported that ICS delivered on the ledge of a jump-up apparatus strengthened avoidance jump-up

responses although it also decreased fear which confirmed similar results reported earlier (Prado-Alcala, Bush, Steele & Reid (1973); Paxton, Mejta & Reid (1974).

Baum, Leclerc and St. Laurent (1973) reported aversive brain stimulation to the pontis caudalis had no effect on RP treatment, while Leclerc, St. Laurent and Baum (1973) reported positive stimulation to the anterior lateral hypothalamus failed to facilitate RP treatment.

A counter-conditioning explanation has been proposed by Metja, et al., (1974) and Reid, et al., (1973) to account for the facilitation of RP treatment on avoidance extinction by ICS.

Social Facilitation

Baum (1969c) reported brief (5 minutes) RP failed to enhance avoidance extinction, but when nonfearful subjects were placed into the apparatus with the experimental subject during RP, RP was found to facilitate avoidance extinction. Baum (1969c) termed this result a social facilitation effect and proposed the occurrence of non-fear behaviour, relaxation, by subjects during RP was necessary for RP treatment to be effective in hastening avoidance extinction.

Reynierse, Klomp and Bach (1974) failed to observe a social facilitation effect. However, this result must by viewed with caution given a methodological weakness of their study, as they themselves state:

> ... the low baseline undoubtedly prevented the "social facilitation" conditions from being statistically effective ... (p. 419).

Corriveau, Contildes and Smith (1978) also investigated the social facilitation effect but used approach methodology

as an assessment of fear rather than reduction in avoidance responding as used by Baum (1969c) and Reynierse et al., (1974). Corriveau et al., (1978) failed to obtain a social facilitation effect, that is, social facilitation did not increase the efficacy of RP treatment. To date, only Baum (1969c) has reported social facilitation increasing the effectiveness of RP. Experiments reported in this thesis also investigated the social facilitation effect using approach methodology as an assessment of fear after Corriveau et al., (1978).

### Miscellaneous Variables Facilitating the Efficacy of Response Prevention

A number of variables have been manipulated which result in increasing the effectiveness of RP to hasten avoidance extinction. These variables include feeding assisted RP (Gale, Sturmfels & Gale, 1966; Moltz, 1954; Nelson, 1966; Sermat & Sheppard, 1959); mechanically forcing the subject to explore the apparatus during RP (Lederhendler & Baum, 1970); providing nesting material to female rats during RP who previously received progesterone (Reynierse & Straw, 1974) and providing distraction noises during RP (Baum & Gordon, 1970).

### Response Prevention in Comparison to other Methods of Facilitating Avoidance Extinction

Thus far we have examined research investigating parametric and procedural variations of the response prevention procedure, per se. A number of studies have also examined the relative efficacy of RP versus systematic desensitization (SD), flooding and regular or massed-trials extinction in hastening avoidance extinction.

## Response Prevention versus Regular Extinction

Numerous studies have shown response prevention to be superior to regular extinction and/or no-treatment controls in hastening avoidance extinction (Bankart, 1972; Baum, 1966, 1971; Black, 1958; Berman & Katzev, 1972; Coulter et al., 1969; Linton et al., 1979; Miller, Mineka & Cook, 1982; Nelson, 1969; Page, 1955; Page & Hall, 1953; Shearman, 1970; Shipley et al., 1971). Delprato and Dreilinger (1974) reported that regular extinction was superior to RP when fear and avoidance extinction was assessed using the passive avoidance procedure. This result was taken to support Denny's (1971) relaxation theory.

A variation of the regular extinction procedure is the massed-trials (MT) extinction procedure whereby the inter-trial interval is reduced markedly. Baum and Oler (1968) found the MT procedure to be more effective than RP in hastening avoidance extinction. Baum and Oler (1968) failed to control non-reinforced CS exposure duration across groups, a factor known to be important in avoidance extinction (Mineka, 1979; Mineka & Gino, 1979b). Blampied and Samuels (1983) controlled non-reinforced CS exposure duration in a replication of Baum and Oler (1968) and reported the MT procedure was superior to RP in hastening avoidance extinction.

## Response Prevention versus Systematic Desensitization

Wilson (1972, 1973) initially found no significant difference between RP and SD in decreasing resistance to avoidance extinction, but when non reinforced CS exposure was controlled in both procedures, RP was more effective in

hastening avoidance extinction. Dickson, Mellgren, Fountain and Dyck (1977) reported no significant difference between RP and a number of SD procedures in reducing approach latencies in a passive avoidance paradigm. Given that only a few studies have compared RP with SD, only tentative conclusions can be drawn. It seems both RP and SD are equally effective in reducing avoidance extinction although further research is required to examine the relative effectiveness of both procedures.

Response Prevention versus Flooding

Polin (1959) reported flooding was significantly more effective than RP in hastening avoidance extinction. However, this result could be due to the flooding subjects receiving one long massed non reinforced CS exposure whereas RP subjects received a series of short CS exposures. A further confound in his experiment was the 5.0 second CS exposure time employed for each RP subject maybe too brief to result in facilitated avoidance extinction (Baum, 1969b). Indeed the opposite effect, enhancement of fear, has resulted from brief non reinforced CS exposure (Coulter et al., 1969; Linton et al., 1970; Rohrbaugh et al., 1972).

Shearman (1970) noted that CS presentation and response availability were confounded in the Polin (1959) study and so controlled these factors in his study. Shearman (1970) reported no significant differences between RP and flooding in hastening avoidance extinction. A finding subsequently supported by Bersh and Paynter (1972) and Baum (1973a). Bankart and Elliott (1974) noted both Polin (1959) and Shearman (1970) used a two-way avoidance procedure. Bankart and

Elliott (1974) replicated the Shearman (1970) design using a one-way avoidance procedure and reported RP was significantly more effective in facilitating avoidance extinction in comparison to flooding. The superiority of RP over flooding has also been reported by Weinberger (1965), Bankart (1972) and Katzev (1972).

In summary, the above results from animal studies indicate both RP and flooding are effective in hastening avoidance extinction but they are inconclusive with regard to which procedure is superior. Indeed, given the focus of attention on these clinical intervention procedures in recent years, it is surprising so few direct comparison studies have been conducted.

Dua (1979) using an analog population (university personnel) compared RP with flooding and reported RP was more effective in facilitating avoidance extinction. He concluded:

> It is clear from the results that the most effective technique of extinction is one that establishes the independence of responding and CS termination by exposure to the fear cues without allowing the subject to respond. (p. 39).

Dua (1979) also reported while RP and flooding facilitated avoidance extinction they failed to extinguish fear. This finding again highlights the dissociation of avoidance and fear extinction, (Mineka, 1979; Morokoff & Timberlake, 1971; Schiff, Smith & Prochaska, 1972; Starr & Mineka, 1977).

Yet the Dua (1979) result should be contrasted with other studies that have found RP effective in hastening both avoidance and fear extinction (Bersh & Paynter, 1972;

Corriveau & Smith, 1978; Monti & Smith, 1976).

In summary, a majority of studies indicate response prevention to be superior to regular extinction in hastening avoidance extinction although in the only studies to directly compare MT and RP, the massed-trials extinction procedure is superior to response prevention.

## Response Prevention: Theoretical Analysis

### Introduction

A number of researchers have proposed that if a theory of avoidance is to be regarded as adequate then it must also explain why response prevention and flooding procedures hasten avoidance extinction. Buss and Reid (1973) exemplified this position by saying:

> The determination of the most efficient ways to reduce persisting avoidance after discontinuation of the aversive stimulation is germane to the development of theories of maintenance of avoidance as well as to therapies for the remediation of phobias and hyperanxiety. (p. 418).

Along similar lines Mineka (1978) stated:

> Flooding or response prevention techniques are effective in producing rapid extinction of well-learned avoidance responses, even though such responses are highly resistant to extinction with conventional extinction procedures ... Any comprehensive theory of avoidance learning must be able to incorporate the findings coming from flooding research and at least be consistent with a satisfactory account of why these techniques are so effective in hastening the extinction of this otherwise persistent behaviour. (p. 1).

Let us examine theories of response prevention and

flooding techniques for hastening avoidance extinction.

Two-Process Theory

According to Mowrer's two process theory, during RP presentation of the CS is no longer paired with the UCS, resulting in fear of the CS extinguishing via the process of Pavlovian extinction. This results also in the elimination of both the motivation and reinforcement of continued responding and so avoidance extinction occurs. Thus, two process theory clearly predicts that as a result of RP treatment both fear and avoidance extinction occurs. However, a number of investigators have noted that while avoidance extinction takes place, fear of the CS sometimes remains following RP treatment (Coulter, et al., 1969; Linton, et al., 1970; Page, 1955; see Riccio & Silvestri, 1973; for a review).

Shipley, Mock and Levis (1971) criticized the above experiments for failing to control total non-reinforced CS exposure across the groups. Shipley et al., (1970), with non reinforced CS exposure controlled across groups, replicated the above studies and reported no difference in residual fear for RP and control groups. They interpreted this result as support for two process theory. It would seem total non reinforced CS exposure is an important variable in producing rapid avoidance extinction, a conclusion also supported by the results of Berman and Katzev (1972).

Another difficulty for two process theory is how to adequately explain why social facilitation, mechanical facilitation and distraction noises increases the efficacy of RP. Possibly two process theory explains this effect by

positing that social and mechanical facilitation procedures bring about an increase in absolute CS exposure which in turn results in facilitated Pavlovian extinction of conditioned fear.

If Pavlovian fear extinction was the only process producing rapid fear and avoidance extinction during non-reinforced CS exposure then subjects which have met the same extinction criterion should have done so because their conditioned fear to the CS extinguished equally, or to put it another way, groups trained to the same extinction criterion should exhibit the same amount of, or lack of, fear to the CS. Also, this should occur whether the response was prevented during RP or allowed to take place during flooding or regular extinction. The evidence indicates that subjects reaching the same extinction criteria do not show equivalent amounts of fear to the CS (Coulter et al., 1969; Linton et al., 1970). It would therefore seem that some other form of learning in addition to Pavlovian fear extinction contributes to rapid avoidance and fear extinction during RP.

While traditional two process theory can explain the majority of RP studies with several studies reporting Pavlovian extinction processes during RP (Shipley et al., 1971; Berman & Katzev, 1972; Bersh & Paynter, 1972; Bersh et al., 1982; Corriveau & Smith, 1978), studies investigating effective methods of facilitating RP or reporting residual fear following RP have proved the Achilles' heel for the two process theory account of RP.

Safety-Signal Theory

Safety-signal theory proposes that extinction of the positive reinforcing function of the avoidance response as a $CS^-$ results during non reinforced CS exposure of RP. With avoidance responding prevented during RP, positive reinforcement no longer takes place in the presence of aversive stimulus cues resulting in avoidance/fear extinction. Safety-signal theory predicts that following RP the avoidance response would no longer function as a positive reinforcer. This prediction has not been tested.

Safety-signal theory has been criticized by Seligman and Johnston (1973) and Mineka (1979). Selgman and Johnston (1973) stated that this theory totally fails to explain RP effects as follows:

> If it is only the positive reinforcement of the response provided by a safety signal which maintains avoidance, and fear has dropped out, there is no fear CR to be extinguished. Since the compound CS is not fear evoking anyway, pairing it with no shock should be irrelevant. The safety signal view is silent on why pairing the external CS and nonresponding with no shock should break up avoidance. (p. 85).

At present, on the basis of the scant research, the safety-signal account of RP appears to be inadequate. Further research focusing on the positive reinforcing properties of the avoidance response as a $CS^-$ are desirable and would enable a more judicious conclusion to be made.

Competing Response Theory

A number of researchers (Page, 1955; Coulter et al., 1969; Linton et al., 1970) have proposed responses other

than the conditioned avoidance response are learned during RP. This learning occurs because residual fear remains during RP which motivates the occurrence of new competing responses, such as freezing or crouching, which are adventitiously reinforced by being paired with the absence of the aversive UCS. The theory asserts further that during subsequent extinction testing the subject exhibits a preference for emitting these newly learned competing responses during RP over the conditioned avoidance response learned during avoidance acquisition training. Research reporting residual fear following RP (Coulter, et al., 1969; Gordon et al., 1979; Linton, et al., 1970; Page, 1955; Page & Hall, 1953; Rohrbaugh & Riccio, 1970) and research showing a dissociation between avoidance and fear extinction (Starr & Mineka, 1977; Mineka, 1979; Mineka & Gino, 1979b) has been taken to support competing response theory.

A number of criticisms have been levelled at the competing response account of RP. Baum (1970) argues as did Kimble (1961) that the source of these competing responses is not specified and crouching and freezing are only two of a number of possible responses exhibited during RP. His own research revealed subjects also exhibited exploratory and relaxation behaviours during RP (Baum, 1969b, c). A difficulty for the theory is to determine reliably a priori, what behaviour response classes subjects will exhibit during RP. Baum (1970) also suggested that competing response theory, as well as two process theory, has difficulty in explaining why certain variables affect the efficacy of RP.

Mineka (1979) argued that competing responses have been shown in a number of studies to occur during RP, but it is

yet to be conclusively demonstrated that such responses mediate or are necessary for avoidance-fear extinction. From the evidence, it would seem that competing responses do not mediate the extinction process, as Mineka (1979) illustrates:

> ... Black's (1958, 1959) results from flooding done under curare seem to show that a learned competing response is not necessary for extinction to occur: Dogs given no opportunity to learn a competing motor response because they were paralyzed by curare when flooding was carried out still showed rapid avoidance response extinction. (pp. 998-999).

Franchina, Hauser and Agee (1975) interpolated a reinstatement procedure, avoidance re-acquisition, between RP treatment and extinction testing. They failed to find reliable differences in performance between RP and control subjects on terminal re-acquisition trials, but RP subjects showed facilitated avoidance extinction relative to controls. They argue this result is difficult for competing response theory to handle, because .....

> If an incompatible response like sitting was established during response prevention ... recurrence of this response ... during retraining would have been punished. Repeated retraining trials would then have provided continuous punishment for responses incompatible with escape and ... provided reinforcement (via shock offset) following escape behaviour. Consequently, retraining procedures should have facilitated the re-emergence of the originally trained escape response and decreased the likelihood of competing behaviour (Bersh and Keltz, 1971). (p. 6).

Bersh, Whitehouse and Mauro (1982) showed both competing responses and Pavlovian fear extinction during RP were implicated in the facilitation of avoidance extinction. While Bersh et al., (1982) demonstrated that a competing response interpretation might be used to explain a number of behavioural procedures such as extinction, punishment, learned helplessness and omission training, they proposed a more rigorous scientific approach was required when using this analysis:

> The present research provides a clear-cut demonstration that competing behaviour can serve the type of function attributed to it ... but, unless its acquisition and its interactions with previously conditioned behaviour or its impact upon attempts to condition new behaviour can be traced explicitly, such theoretical accounts must remain largely speculative (cf. Mackintosh, 1974). (p. 132).

Relaxation Theory

As we have just noted, Baum (1970) criticized the validity of the competing response account of RP, and in the same article proposed an alternative theory, relaxation theory, which in essence, substitutes freezing and crouching behaviour proposed by the competing response theorists for "undifferentiated exploratory behaviour and grooming" (p. 281). Baum's (1970) theory resembles Denny's (1971) relaxation theory. According to Denny (1971) avoidance extinction takes place as a result of the backchaining of relaxation from the safe area of the apparatus and/or the period following UCS offset and termination of UCS associated cues to the CS. Baum (1970) states that during RP subjects learn to relax, a result supported from use of time sampling techniques of

behavioural observations made during RP (Baum 1969b, 1969c). According to relaxation theory, occurrence of nonfearful relaxation behaviours is necessary for RP to be effective in facilitating avoidance extinction.

Delprato and Dreilinger (1974) directly investigated the relaxation theory analysis and reported results consistent with such an analysis. They argued that regular extinction should produce greater backchaining of relaxation than RP, and consequently, regular extinction should be superior to RP in facilitating avoidance extinction. Delprato and Dreilinger (1974) reported this result, but this study stands alone in supporting a superiority of regular extinction over RP among numerous studies comparing the two techniques.

The studies examining the massed-trial extinction procedure (Baum & Oler, 1968; Blampied & Samuels, 1983) have produced results in direct conflict with relaxation theory. As Blampied and Samuels (1983) explain:

> The superiority of massed trials over response prevention ... conflicts with Denny's (1971) relaxation theory, since the 3 seconds safe-area exposure per trial in the massed trials condition was too brief to allow for any relaxation to have occurred there, hence little relaxation from the source could be expected to have "back-chained" or generalized to the CS ... In contrast, response prevention subjects spent 468 seconds on the floor unable to make the response ... it might be expected that relaxational responses would have been well established during the longer response prevention period used in this study. Therefore, if the learning of relaxational responses mediates the reduction of fear and avoidance behaviour it is clear that response prevention, rather than massed trials, should have been the most effective

treatment. (p. 206).

The finding of residual fear following RP although avoidance extinction has occurred is difficult for relaxation theory to explain. In a similar vein, Morokoff and Timberlake (1971) reported that subjects which showed rapid avoidance extinction displayed more fear responses in comparison to subjects which extinguished more slowly, a result in conflict with relaxation theory. Baum (1970) admitted relaxation theory has difficulty in accounting for procedures which increase the efficacy of RP treatment. The foregoing discussion indicates while relaxation responses occur during RP they do not mediate RP facilitated avoidance extinction.

Cognitive Theory

Seligman and Johnston (1973) proposed that during avoidance acquisition, the subject learns two expectancies, firstly, that nonresponding leads to presentation of the aversive UCS and, secondly that responding leads to the absence of the UCS. Response prevention treatment, according to cognitive theory, leads to the disconfirmation and weakening of these expectancies, but conformation and strengthening of the expectancy that non responding does not lead to UCS presentation. Seligman and Johnston (1973) also state:

> Unlike two process theory, our theory asserts that response extinction occurs independently of Pavlovian fear extinction. It is therefore entirely possible according to our account that after response blocking some animals could still be afraid of the CS even though the avoidance response had entirely extinguished. (p. 93).

Cognitive theory does represent a parsimonious explanation of RP facilitated avoidance extinction. It explains any outcome from an RP experiment by alluding to confirmation or disconfirmation of a particular expectancy by the subjects. Mineka (1979) notes that this also poses a problem for the theory by taking it into the realms of untestability. Along similar lines Marks (1977) had this to say regarding cognitive interpretations in psychopathology,

> It is so easy to equate fear of snakes with fear of the penis, or impotence with castration anxiety: validating such an equation is a task fraught with difficulties. Even when the fantasies are clearly those of the patient rather than of the therapist, the question remains whether they are the product or the cause of his psychopathology ... This chicken and egg issue often bedevils interpretation of cognitions in psychopathology. (p. 206).

SSDR Theory

A number of investigators have applied a species specific defense reaction (SSDR) analysis to RP research (Bersh & Keltz, 1971; Crawford, 1975, 1977; Mineka & Gino, 1979a; Mineka, Miller, Gino & Giencke, 1981). SSDR theory proposes during active avoidance acquisition, incompatible responses such as freezing are punished, however, during RP with the active avoidance response prevented, another SSDR, freezing, occurs and is maintained by the absence of the aversive UCS. Following RP treatment, the dominant SSDR during extinction testing is freezing. Crawford (1977) makes the distinction between the non-associative SSDR theory and associative competing response theory as follows:

> The competing-response hypothesis specifies that

> freezing is learned, subject to the same reinforcement contingencies that lead to the learning of the original avoidance response. The SSDR hypothesis, on the other hand, assumes that freezing is an innate SSDR which will spontaneously appear in dangerous situations. The active-avoidance contingencies serve to suppress freezing by punishing it with shock; response prevention serves to increase the probability of freezing by making the flight SSDR impossible. (p. 40).

Mineka (1977) reported brief confinement in a novel place following avoidance acquisition facilitates avoidance extinction. She argues this result is best explained by SSDR theory, while being inconsistent with two process and competing response theories.

Mineka (1979) believed that although Crawford (1977) demonstrated that a change in SSDR hierarchy produced RP effects, she failed to show that RP normally acts through such a process. She maintained that such an effect maybe temporary and if a time delay between brief confinement in a novel or fearful place and subsequent extinction testing had been included, then brief confinement may have resulted in no effect.

Mineka (1976) reported RP with one avoidance response facilitated extinction of another avoidance response learned with a different CS. She termed this the irrelevant flooding effect and proposed neither competing response, relaxation nor cognitive theory can explain this effect. Possibly SSDR theory can, by positing that the dominant SSDR is changed from flight to freezing by the irrelevant RP procedure. In assessing this possibility, Mineka (1979) concluded

insufficient evidence was available to make a firm statement in favour of the SSDR analysis of the irrelevant RP effect.

Mineka and Gino (1979b) further investigated the brief confinement effect in a novel place (Crawford, 1977) and found the procedural requirement of starting jump-up avoidance extinction on the grid-floor produced the confinement effect, while starting on the safety ledge reduced the effect. They also reported a failure to obtain the confinement effect with a well-trained jump-up avoidance response procedure. In conclusion, Mineka and Gino (1979b) stated:

> Thus we maintain that there is little reason ... to believe that confinement in novel or fearful places produces a robust phenomenon comparable to that produced by flooding ... Because of the inherently weak nature of this effect, it cannot provide compelling support for the SSDR theory of flooding. (p. 113).

Effective Reinforcement Theory (ERT)

To date, ERT has not been formally proposed as an explanation of RP treatment, yet it would seem to be applicable. ERT apportions conditioned fear to both the CS and the situational cues of the avoidance procedure, while two-process theory focuses on conditioned fear of the CS. ERT explains RP-facilitated avoidance extinction by proposing that during RP non-reinforced exposure occurs to both the feared CS and feared situational cues. A RP procedure which maximally reduces both sources of conditioned fear would produce rapid avoidance fear extinction. RP procedures which increase non-reinforced exposure to situational cues, such as mechanical facilitation (Lederhendler & Baum, 1970),

social facilitation (Baum, 1976b) and observational facilitation (Uno, Greer & Goates, 1973), have increased the efficacy of RP in facilitating avoidance extinction, thus supporting ERT.

Franchina, Agee and Hauser (1974) gave RP in either the avoidance acquisition apparatus or a wire cage acting as a dissimilar environment to the avoidance apparatus. They reported RP was more effective in facilitating avoidance extinction when conducted in the avoidance apparatus in comparison to the wire cage. This finding supports ERT. While support for ERT is found in the RP literature, more research is required focusing on the elimination of conditioned fear to both the CS and situational cues.

Incubation Theory

Incubation theory (Eysenck, 1967, 1968, 1976, 1979) has focused on fear-avoidance extinction rather than representing a comprehensive analysis of fear-avoidance acquisition and extinction. Yet it has generated much interest and recent research. It is also applicable to RP effects on avoidance extinction, although other theories of fear enhancement do exist (Gordon et al., 1979; Miller & Levis, 1971; Watts, 1979). Incubation theory represents an attempt to explain fear-avoidance enhancement following non-reinforced CS exposure (Rohrbaugh & Riccio, 1970; Rohrbaugh et al., 1972; Silvestri et al., 1970).

Woods (1974) summarizes the main points of incubation theory as follows:

> Eysenck suggested that after conditioning with a painful UCS, unreinforced CS occurrences

> may be expected to produce incremental as well as decremental changes in response strength, observed CRs being the resultant of these two opposing influences. Decremental changes may be expected as the result of normal extinction processes. Incremental effects on the CR as a result of CS- only presentation are accounted for by the fact that an average CS is followed by painful autonomic effects with real UCR properties. These secondary UCRs may be thought of as response-produced stimuli and come to evoke more fear, producing a positive feed back mechanism reinforcing the original CS-CR association. Usually the extinction process will be stronger than this reinforcement effect, and the CR will gradually reduce in strength. In cases of extremely strong UCS and short durations of CS exposure, however, incremental influences may predominate, with a consequent enhancement of CR. (pp. 300-301).

Eysenck (1979) proposed that CR strength is a negatively accerelating function of increasing the non-reinforced CS exposure duration. In other words, small variations in short CS exposure durations produce large magnitude changes in CR strength, while small variations in long CS exposure durations produce relatively small magnitude changes in CR strength (cf. Eysenck, 1979; figure 1; Bersh, 1980; figure 1). Points along this negatively accerelating curve represent critical values of the joint effect of CR strength as a function of non-reinforced CS exposure duration. Points above the curve represent an incubation effect, points below an extinction effect. Flooding and RP, therefore, result in avoidance extinction by decreasing CR strength, for a given CS exposure duration, resulting in a joint value of CR strength and CS exposure duration below the critical points.

That is, flooding and RP move the negatively decelerating curve downwards. Fear enhancement is explained by the critical value being exceeded during RP or flooding, primarily because CS exposure was too brief. Incubation theory also predicts that increasing UCS intensity, thereby increasing CR strength, necessitates increasing non-reinforced CS exposure duration to obtain extinction rather than incubation. Results investigating UCS intensity on RP discussed earlier seem to indicate this to be the case (Baum, 1969a, b, 1970; Sielgeltuch & Baum, 1971), although Ward (1976) failed to find this relationship.

Levis (1979) provided as critique of incubation theory and in doing so proposed fear enhancement as explanable in terms of modified two process theory (Levis & Boyd, 1979; Stampfl & Levis, 1976) as follows:

> ... that extreme resistance to extinction is a function of short-latency avoidance responses preventing the extinction of fear to longer CS exposures and eliminating the ability of longer CS exposures to recondition short latency responses. Thus the secondary intermittent conditioning effect produced by longer exposures to the CS is believed responsible for enhancing avoidance maintenance. The enhancement of fear to the CS in extinction (incubation) is explained by an increase in exposure to the nonextinguished part of the CS chain which can be dramatic especially if serial CS cues are used. (p. 173).

A number of studies have now investigated predictions derived from incubation theory. To date some studies have failed to replicate the fear enhancement effect (Kaloupek, 1983; Kaloupek, Peterson, Boyd & Levis, 1981; Nicholaichuk, Quesnel & Tait, 1982) while others support incubation theory

(Boyd, 1981; Miller & Levis, 1971; Stone & Borkovec, 1975). At present, it would seem premature to accept or reject incubation theory as an explanation of fear enhancement following brief non-reinforced CS exposure.

In summary, all the theories of RP that have been discussed explain some of the RP research, but not all, and all theories can be regarded as being partially supported by the relevant research. However, in making such a blanket statement it is important to note that RP research has been conducted with a variety of species (animal and human), with different response tasks trained to different criteria. Such diversity of experimentation makes global comparison of theories a very dangerous task. This together with the possibility that different learning processes occur with different response tasks and fear extinction being more central to avoidance extinction of particular avoidance responses in comparison to others, caution is warranted in making unequivocal assertions regarding the explanatory power of one particular theory over another (see Mineka, 1979).

## Fear Assessment Techniques

The majority of the research which has just been reviewed found prolonged RP treatment facilities fear-avoidance extinction when fear assessment is by persistence of avoidance responding during extinction testing (Baum, 1968a, b, 1969a, 1970; Berman & Katzev, 1972; Corriveau, 1978; Coulter, et al., 1969; Mineka & Gino, 1979a) or the CER assessment techniques after Estes and Skinner (1941), (Bankart and Elliott, 1974; Monti & Smith, 1976; Mineka & Gino, 1979b; Starr & Mineka, 1977) and approach assessment

methodologies (Bersh & Paynter, 1972; Corriveau, 1978; Corriveau & Smith, 1978; Corriveau et al., 1978; Mineka et al., 1981).

Typically most investigations of RP effects have used only one fear assessment measure when it is widely known there is often a lack of concordance among the assessment procedures therefore indicating multiple response measures should be taken (Lang, 1968; Mineka, 1979). Another issue is the use and interpretation of the fear construct. The covert nature of this mediating variable has proved disagreeable to a number of operant theorists (Herrnstein; 1969; Herrnstein & Hineline, 1966, Hineline, 1977). Yet a purely operant analysis of avoidance procedures fails to adequately account for acquired drive properties of avoidance responding (Bolles, 1975a, 1975b).

The foregoing discussion on RP associated research and theory indicated the fear construct probably needs to be retained, with alternative theories placing different emphasis on the role of fear extinction, whether it is a necessary or sufficient condition for avoidance extinction. Indeed the situation was found to be extremely complex.

Traditionally, reduction of condtioned avoidance responding in the absence of UCS presentation was taken to reflect fear extinction. The majority of RP studies follow this traditional line by employing avoidance response reduction as the dependant variable in assessing RP effects. A number of studies using this procedure by incorporating RP treatment between avoidance acquisition and extinction have failed to provide adequate controls for either CS duration during RP or exposure to apparatus cues for the

same duration as RP or both (Baum,1966, 1969a; Baum & Higgins, 1971; Lederlendler & Baum, 1970; Linton et al., 1970; Page, 1955; Page & Hall, 1953; Siegeltuch & Baum, 1971).

The approach latency assessment procedure was first developed by the competing response theorists (Page, 1955; Coulter et al., 1969; Linton, et al., 1970). They argued, from their competing response position, that their procedure was more valid in assessing RP effects as the traditional method could produce artifactual results. Shipley, Mock and Levis (1971) criticized the Page (1955) and Coulter et al., (1960) studies on the following grounds:

> At the time of the fear test, the regular extinction and response delay groups ... had received more CS exposure than the response prevention groups ... with CS exposure held constant, two other factors ... can vary: (a) the number of times subjects are transported from the nonshock side to the shock side of the apparatus and (b) the amount of exposure to apparatus cues of the nonshock and shock sides ... during the intertrial interval. (p. 257).

To control CS exposure, exposure to apparatus cues and trials to extinction Shipley et al., (1971) eliminated avoidance extinction testing following RP treatment and instead immediately tested subjects using approach methodology procedures. As Corriveau and Smith (1978) point out, the Shipley et al., (1971) study had the methodological weakness of failing to present CS cues during approach latency testing. The present thesis further investigates the approach methodology as a fear assessment technique.

A further point arising from the Page et al. studies

needs to be raised. While it is still possible for fear extinction to take place as a result of non-reinforced CS exposure, the use of approach latency as a measure of fear assessment highlights the problem of using the reduction of avoidance responding in an active avoidance extinction procedure as a valid index of fear. A number of investigators have shown that approach assessment techniques provide a more sensitive measure of fear than the traditional reduction in avoidance responding method (Corriveau;, 1978; Corriveau & Smith, 1978; Mineka, et al., 1981; Miller, Mineka & Cook, 1982). Indeed the approach latency procedures can be regarded as either one-way passive avoidance (Bersh & Paynter, 1972; Shipley et al., 1971), or step-down passive avoidance (Mineka et al., 1981; Miller et al., 1982) with both passive avoidance procedures incorporating safety-test behaviours (Baum, 1969; Corriveau & Smith, 1978; Spring et al., 1974).

Another method to assess fear-anxiety comes from the conditional emotional response (CER) paradigm initially developed by Estes and Skinner (1941). The typical CER procedure consists of presenting a previously avoided CS or a Pavlovian fear CS, independent of the subject's behaviour, during the course of ongoing operant appetitive responding. Appetitive response suppression during CS presentation is taken as an indication of conditioned fear to the CS. Greater suppression is assumed to represent greater conditioned fear-anxiety to the CS. Research has indicated CER procedures are more sensitive to fear extinction than avoidance response reduction procedures (Monti & Smith, 1976; Mineka & Gino, 1979b).

While all fear assessment methodologies discussed have conceptual advantages and disadvantages (Corriveau & Smith, 1978; Corriveau et al., 1978; Monti & Smith, 1976; Testa, 1976) the CER procedure seems to be particularly sensitive to the autonomic signs of fear-anxiety. Given this sensitivity of the CER procedure to these aspects of fear and anxiety the present study used the CER procedure to examine the anxiolytic action of benzodiazepine and beta-adrenergic blocking agents as suggested by Blampied and Kirk (1983).

In the present study, benzodiazepine and beta-adrenergic blocking agent action was examined using different fear assessment methodologies, the approach methodology and the CER procedure. Making empirical comparisons between various fear assessment methodologies allows investigation of the following questions: are the results of the different fear assessment procedures comparable? Are they measuring the same or different attributes of the fear-anxiety construct? Are they equally or differentially sensitive or powerful as dependant variables? Investigation of anxiolytic drug action was either incorporated into a design investigating RP effect on avoidance and fear extinction or was investigated in its own right.

In summary, this literature review has highlighted a number of persistent issues in avoidance research: 1. the role of the CS and US in avoidance behaviour; 2. the determination of the effective reinforcer in avoidance behaviour; 3. the high resistance to extinction of avoidance behaviour; 4. the role of the avoidance response, non-associative processes, in avoidance behaviour; 5. the role of fear and response-outcome expectancies in the

acquisition, maintanance and extinction of avoidance behaviour; 6. the association/dissociation of fear and avoidance behaviour; and 7. the similarities between methods which hasten avoidance extinction and those used to treat fear motivated human neurotic behaviours. This thesis examines those issues which pertain to the extinction of fear and avoidance behaviour with the view that further elucidation of the determinants of fear extinction during and following RP treatment will facilitate an understanding of the role of fear in avoidance extinction and also its role in behaviourally based therapeutic approaches to fear motivated human neurotic behaviours.

# CHAPTER TWO

## Escape from Fear, Response Prevention and the Development of an Approach Method for Fear Assessment

## EXPERIMENT: ONE

### Escape from Fear: Replication and Response Prevention Effects.

### Introduction

A common problem with traditional fear assessment procedures is that the behavioural measures by which the subject's fear is measured show a lot of between and within-subject variability. This makes it difficult to use these measures to assess the effects of drugs or other putative treatments on conditioned fear (Mineka, 1979). The escape from fear (EFF) paradigm offers a possible solution to these difficulties while at the same time providing a baseline to assess response prevention effects on conditioned fear. Because conditioning of fear is separate from learning the instrumental escape response in the EFF procedure, it is possible to independently examine drug effects on these two phases of the EFF procedure.

The typical escape from fear procedure is described by McAllister, McAllister, Hampton & Scoles (1980) as follows:

> In a typical escape-from-fear (EFF) task, classical fear-conditioning trials (CS + shock) are administered in one side of a two-compartment apparatus. Later, in the absence of shock, subjects are allowed to jump a hurdle to an adjacent safe

> compartment and, thereby, to escape the fear eliciting CS and situational cues in the shock compartment. (p. 417).

Fear associated with both the CS and contextual cues of the shock compartment motivate the subject to escape from this chamber to an adjacent chamber. Reinforcement for the hurdle jump response is provided by fear reduction following the response and CS offset, a two process theory explanation.

The acquired drive or EFF procedure was first used by Miller (1948) and has subsequently been developed by Brown and Jacobs (1949); Desiderato, (1964); Desiderato, Butler and Meyer, (1966); Goldstein (1960, 1965, 1974, 1976a, b, c); McAllister and McAllister (1962a, b, 1963, 1965, 1967, 1971); McAllister, McAllister, Brooks and Goldman (1972); McAllister, McAllister, Weldin and Cohen, (1974); and McAllister, McAllister, Hampton and Scholes (1980).

A number of variables have been investigated using the EFF procedure; these include the delay interval between classical fear conditioning and operant learning (McAllister & McAllister, 1962a, 1963, 1965, 1971; Desiderato, Butler & Meyer, 1966; Kent, Wagner & Gannon, 1960; Wolz, Hurst, Sherr & Spear, 1979); the number of CS-UCS pairings during classical fear conditioning (Desiderato, 1964; Goldstein, 1960, 1976c; Gwinn, 1951; Kalish, 1952, 1954); manipulating UCS intensity (Goldstein, 1960; McAllister & McAllister, 1962a; Miller & Lawrence, 1951); varying the CS-UCS interval (Crawford, Masterson & Wilson, 1977; McAllister, McAllister, Weldin & Cohen, 1974); altering CS intensity or presence (Desiderato, 1964; McAllister et al., 1974); neurophysiological effects (Goldstein, 1965;

King & Cairncross, 1974); varying CS-US contingency (Crawford et al., 1977) and investigating drug effects on performance (Coover, et al., 1978; Cunningham & Brown, 1983; Daly, 1968).

A number of investigators have reported good EFF performance with a 24 hour delay between conditioning and testing (Goldstein, 1960; Kalish, 1954; McAllister & McAllister, 1962b), poor performance with a 90 minute delay (Desiderato, 1964) and little or no escape-from-fear responding when testing was administered immediately following classical fear conditioning (McAllister & McAllister, 1962a).

To account for this delay between conditioning and testing effect McAllister and McAllister (1963) proposed three possible hypotheses:

(a) that fear simply increases with time, an "incubation" effect (McAllister & McAllister, 1967);

(b) that incompatible responses, such as, freezing and crouching, weaken over time; and

(c) because subjects in the McAllisters' laboratory are tested in a fear conditioning box different from, but "constructed to appear identical with" (McAllister & McAllister, 1962a, p. 111) the startbox of the escape-from-fear apparatus, the McAllisters' state that superiority of EFF performance found in the 24 hour delay subjects compared to the no delay subjects was due to the stimulus generalization gradient of fear between the two situations flattening out with time. This is termed the stimulus generalization decrement hypothesis (McAllister & McAllister, 1963, 1965, 1967, 1971; Wolz, Hurst, Sherr & Spear, 1979).

Desiderato, Butler and Meyer (1966) investigated these

three hypotheses by manipulating the delay interval (0, 2, 6, 12 or 24 hours) following 35 CS-UCS pairings. Also for half the subjects in each delay condition the apparatus cues were altered between conditioning and testing. Desiderato, et al., (1966) failed to support hypotheses (a) and (b) above, but reported support for the stimulus generalization decrement hypothesis.

In the present experiment a 24 hour delay occurs between conditioning and testing with the chamber in which subjects received CS-UCS pairings also acting as the startbox for escape-from-fear trials.

The method of handling subjects during conditioning and testing phases of an EFF procedure has varied across studies.

Crawford et al., (1977); Goldstein (1960, 1976c); McAllister and McAllister (1963, 1965, 1967) and McAllister et al., (1980) all placed their subjects into the conditioning chamber at the beginning of the classical fear conditioning session and removed them at the completion of conditioning. Other researchers have removed subjects from the conditioning chamber after each CS-UCS pairing and placed them back immediately prior to the next pairing (Grelle & James, 1981; Spear et al., 1980; Wolz et al., 1979). McAllister et al., (1980) reported the Crawford et al., (1977) procedure of handling subjects by the tail resulted in poorer EFF performance in comparison to their laboratory procedure of handling subjects by the body. In keeping with this result, all subjects in the present experiment were handled by the body. The present experiment involved first establishing escape from fear performance, then examining the effect of response prevention on extinction of EFF performance. In

addition, both male and female subjects were used so as to examine any possible gender effect on EFF performance.

## Method

### Subjects

The subjects were 30 naive male and female New Zealand random bred Wistar rats from the Psychology Department's breeding colony. Their age range at the time of testing was 173-240 days.

Subjects were reared in group housing of 4-5 single sex animals per cage. Subjects were maintained on a 12 hour light / 12 hour dark cycle with all testing conducted during the dark phase of the cycle. All subjects were provided with ad-libitum food and water.

### Apparatus

The apparatus used for Pavlovian fear conditioning and instrumental escape from fear acquisition consisted of two Lafayette A550 modular testing units joined together to form two compartments, the total size being 61.0cm x 25.0cm x 28.0cm (L x W x H). Each compartment had a floor of 18, 0.5cm diameter stainless steel rods spaced at 1.7cm centers. All sides of both compartments were aluminium construction, except the front which had a 1-way window to allow observation of the subject. During habituation and EFF acquisition a stainless steel partition with a 12cm x 9cm (H x W) opening elevated 3cm above the chamber floor was placed between the chambers. During Pavlovian fear conditioning this partition was replaced by a solid stainless steel

partition. A 24v lightbulb positioned immediately above the ceiling of each chamber, permitted independent illumination. One compartment was used for Pavlovian fear conditioning and as the start chamber for EFF acquisition. The other chamber was used as the goal chamber in EFF acquisition and differed in three features from the start chamber, namely, the overhead illumination remained off during EFF acquisition, a stainless steel sheet was placed over the grid rod floor, and the end wall was covered with heavy cardboard having 2.4cm wide horizontal black and white alternating stripes painted on it. These additional features were added to the goal chamber to make it contextually different from the start chamber as the EFF procedure is normally conducted in two contextually different chambers (McAllister, McAllister, Hampton & Scholes, 1980).

The aversive unconditioned stimulus, shock, used during Pavlovian fear conditioning was scrambled through the grid rods of the start chamber using a Laffayette neon grid scrambler (Model 58020) linked to a Lafayette shock source (Model 82400). The nominal setting for shock intensity was 0.5mA. The conditioned stimulus was provided by a Lafayette Sonalert tone generator (Model 58025) which produced a 2800Hz tone when activated. The CS added 10dB (A) to 65dB (A) background white noise used to mask extraneous sounds. Programming of experimental events and recording outcomes was achieved using Pye Hi-Logic solid state and electro-mechanical programming equipment.

## Procedure

The procedure involved four consecutive phases:

1. Habituation and Pavlovian fear conditioning. On day one, subjects explored the apparatus, with access to both chambers, in groups (N = 5) for 15 minutes. Subjects were then removed to a holding cage while a solid stainless steel partition was inserted between the chambers. Each subject was then individually placed into the goal chamber for 30 minutes and then immediately placed into the start chamber for nine Pavlovian fear conditioning trials. Nine CS-UCS pairings were administered with a variable interval 180 second interval between trials. Each trial began with the onset of the tone CS followed 4.0 seconds later by a 1.0 second, 0.5mA inescapable shock. The CS and UCS terminated together. These Pavlovian fear conditioning parameters were chosen following pilot study investigation, being congruent with the contingency analysis of fear conditioning and being ethically desirable in keeping shock exposure to a minimum.

2. Escape-from-fear acquisition. On day two, the partition with an opening was inserted between the compartments. All subjects received 50 hurdle jump EFF trials, without shock, with an ITI of 30 seconds. Each EFF trial began with the subject being placed into the start chamber. Depression of the grid rod floor activated a microswitch which initiated both the tone CS and clock which recorded EFF hurdle jump response latency in seconds (1/100). If the subject performed a hurdle jump response, depression of the goal chamber floor activated a microswitch which terminated both the CS and clock timer. The subject remained in the goal chamber for a 30 seconds (ITI)and was then immediately replaced back into the start chamber for the next EFF trial. If no hurdle jump response occurred within 60.0 seconds of CS

onset, the CS was automatically terminated and a 60.0 second "response" latency was recorded.

3. Response Prevention treatment. On day three, the solid stainless steel partition was inserted between the chambers. Equal numbers (N = 5) of male and female subjects were randomly allocated to one of three of the following groups, (1) Group No-RP controls remained in their home cages; (2) the RP treatment subjects were individually placed into the start chamber ( Pavlovian fear conditioning chamber ) and received either 100 CS presentations (Group RP-100) with a mean variable interval of 30.0 seconds between each CS presentation or (3) 200 CS presentations (Group RP-200) with a mean variable interval of 15.0 seconds between each presentation. The duration of RP treatment session was 50.0 minutes for Group RP-100 and 55.0 minutes for Group RP-200.

4. Escape-from-fear acquisition. On day four, all subjects received a further 50 hurdle jump EFF trials according to the procedure in operation during day two EFF acquisition again without any shocks.

## Results and Discussion

All subjects learned the escape from fear hurdle jump response on day two. The response speed increased from 0.434 $sec.^{-1}$ for the first five EFF trials to 0.946 $sec.^{-1}$ for the last five EFF trials. This represented a statistically significant increase across all subjects ($F(9,261) = 4.56$, $p < .0001$). The EFF hurdle jump response acquisition replicates the McAllister et al.'s (1980) NEP-B group EFF performance.

If RP treatment on day three decreased fear to the CS and contextual cues associated with the classical fear conditioning chamber, then RP subjects should perform the EFF response task on day four with longer latencies in comparison to home cage (day three) controls. As increasing non reinforced CS exposure duration increases the efficacy of RP treatment in hastening fear and avoidance extinction, it was expected that subjects exposed to 200 CS presentations would exhibit longer EFF responses in comparison to Group RP-100 and Group No-RP subjects.

To examine RP treatment effects on day four EFF performance, a three way, Groups x Gender x EFF Trial Blocks, analysis of covariance was computed. Day two EFF performance acted as a covariate for each subject in this analysis. Only the EFF trial blocks Main Effect was significant, ($F(9,215) = 4.84$, $P<.0001$); reflecting a slight decrease in response speeds from the first five EFF trials to the last five EFF trials. As EFF testing continued some extinction of the EFF response took place. This can be seen in Table 2-1 which presents the mean EFF response speeds for female and male subjects in each treatment group across EFF trial blocks.

The expectation that females would perform the EFF response with shorter response latencies was not confirmed by the present experiment (cf. Archer, 1975; Crawford et al., 1977).

The expectation that RP treatment would reduce conditioned fear to the CS and contextual cues of the classical fear conditioning chamber as evidenced by longer EFF response latencies on day four was not confirmed by the present

TABLE 2-1

Mean speed ($sec^{-1}$) of EFF performance for male and female subjects across trial blocks for each treatment group.

| | Gender | | | | | | |
|---|---|---|---|---|---|---|---|
| | Male | | | Female | | | |
| | Treatment Groups | | | | | | |
| Trial Blocks | No RP | RP-100 | RP-200 | No RP | RP-100 | RP-200 | Overall |
| 1 | 0.508 | 0.217 | 0.309 | 0.369 | 0.281 | 0.466 | 0.358 |
| 2 | 0.270 | 0.085 | 0.189 | 0.147 | 0.083 | 0.261 | 0.172 |
| 3 | 0.271 | 0.132 | 0.195 | 0.080 | 0.078 | 0.340 | 0.182 |
| 4 | 0.165 | 0.068 | 0.188 | 0.096 | 0.118 | 0.437 | 0.179 |
| 5 | 0.219 | 0.108 | 0.237 | 0.106 | 0.077 | 0.267 | 0.169 |
| 6 | 0.261 | 0.155 | 0.226 | 0.169 | 0.085 | 0.263 | 0.193 |
| 7 | 0.269 | 0.165 | 0.261 | 0.176 | 0.126 | 0.249 | 0.208 |
| 8 | 0.431 | 0.241 | 0.100 | 0.111 | 0.156 | 0.308 | 0.224 |
| 9 | 0.465 | 0.215 | 0.162 | 0.162 | 0.303 | 0.298 | 0.267 |
| 10 | 0.359 | 0.206 | 0.137 | 0.140 | 0.179 | 0.243 | 0.211 |
| Overall | 0.322 | 0.159 | 0.200 | 0.156 | 0.148 | 0.313 | |

experiments. From Table 2-1 it can be seen that RP-treated male EFF response speeds are slightly slower while RP-treated female EFF response speeds are slightly faster in comparison to control subjects. These differences are only small and failed to reach statistical significance.

Although RP-100 subjects remained in the Pavlovian fear conditioning chamber for 50 minutes RP, non-reinforced CS exposure duration was 8.33 minutes (100 × 5 sec. CS presentations). Similarly, non-reinforced CS exposure for RP-200 subjects amounted to only 16.67 minutes of the 55 minute RP session. It is possible that non-reinforced CS exposure duration was not sufficient to allow for the learning of the extinction contingency. Also CS presentations were only 5.0 seconds in duration, brief enough to generate an incubation effect (Esyenck, 1968). It is possible that the EFF results can be explained as a type of incubation effect?

Eysenck (1968) initially proposed that under conditions of intense UCS delivery during avoidance acquisition which produced a strong, intense CR, followed by brief duration non-reinforced CS exposures, an enhancement of fear rather than extinction would result (see introduction for further discussion).

Gordon, Smith and Katz (1979) proposed an alternative explanation of the incubation effect in terms of a memory reactivation model. Boyd (1984) summarized the model as follows:

> ... that enhancement effects occur when a cueing of old learning precedes, in time, the organisms learning of a new set of contingencies - Pavlovian extinction. Short CS exposures in extinction procedures ... should provide an opportunity for

> cueing to occur first, thereby resulting in enhanced performance due to strengthening memory. At more sustained unreinforced CS exposure durations, the learning of a new contingency (e.g. extinction) would be expected to cancel the effects of incubation... (pp. 31-32).

Gordon et al., (1979) further proposed at short retention intervals between avoidance acquisition and non-reinforced CS exposure, avoidance conditioning memories are readily available to the subject so that further CS exposure would lead quickly to the learning of the extinction contingency. With longer retention intervals, avoidance conditioning memories are theoretically considered to be less accessible with the result that the same duration of CS exposure which produced extinction at short intervals, will only reactivate the avoidance conditioning memories resulting in enhancement of fear, the incubation effect. At longer retention intervals, longer duration CS exposure is required for the subject to learn the extinction contingency. It is also expected that at moderate duration CS exposures, they would have little or no effect because of the opposing effects of incubation and Pavlovian extinction cancelling each other out.

Considering the EFF results, Eysenck's explanation of the incubation effects rests on the premise that intense, aversive CRs interfere with Pavlovian extinction, while Gordon et al. argue reactivation of avoidance conditioning memories interferes with Pavlovian extinction.

In the present experiment, only a moderate intensity UCS was used in the Pavlovian fear conditioning phase. It is doubtful that such a UCS fulfils the requirements of

Eysenck's strong, intense UCS, necessary for the development of strong and aversive CRs. It would seem Eysenck's interpretation of the incubation effect fails to explain the results.

Gordon et al., (1979) proposed with long retention intervals (e.g. 24 hours) and brief CS exposures (e.g. 5secs.) then an incubation effect could result. The results of the present study do seem compatible with the expectations of the memory reactivation model of incubation. The reason why neither incubation nor extinction was illustrated in the present experiment might be that 100 and 200 CS exposure presentations represented a moderate duration of CS exposure, thereby resulting in the opposing effects of incubation and Pavlovian extinction cancelling each other out.

To obtain extinction of conditioned fear and avoidance this experiment could be replicated with the modification of increasing the cumulative duration of non-reinforced CS exposure during the RP treatment session. By increasing the duration of non-reinforced CS exposure and obtaining conditioned fear and avoidance extinction, then this result would be amenable to both two-factor theory and effective reinforcement theory interpretations (see introduction). Future research should examine this possibility.

This experiment failed to confirm the belief that the EFF paradigm as a fear assessment procedure would reveal RP treatment effects on conditioned fear extinction. While this was disappointing, other researchers have also encountered perplexing results when using the EFF paradigm. As Crawford, Masterson and Wilson (1977) noted:

> ... systematic efforts by three researchers over a period of 2 years to investigate many variables which might affect learning in escape-from-fear situations resulted in consistent failure to produce anything that described as "learning-a-new-response-which-is-reinforced-by-CS-offset." (pp. 67-68).

Given the Crawford et al. (1977) experience and the results of the present experiment it was decided not to persue use of the traditional EFF paradigm but rather to modify the procedure to allow fear assessment using the approach methodology technique.

# EXPERIMENT TWO

## Response Prevention Treatment of Extinction of Passive Avoidance: An Approach Method as a Fear Assessment Technique

### Introduction

Several researchers have demonstrated passive avoidance procedures, including measures such as time taken to approach a previously avoided CS complex and total time a subject voluntarily chooses exposure to a feared CS complex provide a very sensitive measure of conditioned fear (Corriveau, 1978; Corriveau & Smith, 1978; Mineka et al., 1981; Miller et al., 1982). It has also been demonstrated with RP treatment of protracted duration that conditioned fear, when assessed using approach procedures, is significantly reduced (Corriveau & Smith, 1978; Miller et al., 1982).

The major advantages of the approach procedure as a fear assessment technique are summarized by Corriveau and Smith (1978) as follows:

> (a) The approach procedure circumvents the problem of using artifically motivated behaviour as an indicant of fear. (b) The entire procedure permits presentation of the entire CS complex intact, as it was during avoidance training. (c) Since a discrete CS is not employed, the basic parameter of response prevention duration is less complex (various combinations of CS durations and CS presentation frequencies need not be introduced). (d) Conceivable conditioning of "competing responses" during response prevention does not contaminate the interpretation of approach behaviour. (e) Subject-selection bias is not introduced. (p.155).

A purpose of experiment two was to further examine the usefulness of an approach procedure as a fear assessment

technique.

While Corriveau and Smith (1978) proposed a discrete CS was unecessary when using the approach procedure, our pilot study results indicated a one-way passive avoidance procedure lead to uncertainty over the designated "safe" and "danger" compartments. This had the consequence of producing high inter-subject variability. This variability was decreased by adding discrete CSs associated with the 'safe' and 'danger' compartments, thus making the compartments more discriminable.

Shipley, Mock and Levis (1971) criticized early approach fear assessment procedures for failing to control CS exposure, exposure to apparatus cues and trials to extinction across groups. To circumvent these problems, Shipley et al., (1971), suggested commencement of the fear assessment procedure immediately following RP treatment and therefore eliminating extinction trials altogether. Both Corriveau and Smith (1978) and experiment two of this study eliminated extinction trials between RP treatment and fear assessment.

Monti and Smith (1976) together with Testa (1976) argued some early studies using the approach methodology confounded the motivational basis for approaching the feared CS by also directly manipulating the food deprivation level of subjects (e.g. Spring, 1973). The question is then asked, did the subjects approach the aversive CS complex because they had access to food or because extinction of fear had occurred?

To overcome this possible confounding effect, in experiment two the animal's exploratory tendency is used as the motivational basis for approach rather than some

explicit deprivation procedure.

Another feature of some studies using the approach methodology to assess fear is incomplete elimination of fear has been reported following RP treatment. To reduce residual fear to a minimum a protracted duration of RP treatment was used in experiment two.

Another purpose of this experiment is to further examine the effects of massed vs distributed RP treatment on subsequent fear and avoidance extinction using the approach assessment technique. Animal studies investigating the efficacy of massed vs distributed RP have produced inconsistant results. Polin (1959) reported massed RP was superior to distributed RP in hastening avoidance fear extinction. Superiority of distributed RP over massed RP has also been reported (Baum & Myran, 1971; Berman & Katzev, 1972; Franchina, Agee & Hunter, 1974), while still other researchers have reported no significant difference in the effects of massed vs distributed RP (Bankart & Elliott, 1974; Shearman, 1970; Shipley, 1974).

In the human literature a consistant finding is the superiority of massed RP over distributed RP (Mathews & Shaw, 1973; Stern & Marks, 1973; Chaplin & Levine, 1981; Foa & Chambless, 1978).

A major difference between the fear assessment phase of Corriveau and Smith (1978) and the present study is the duration of fear assessment. Whereas the Corriveau and Smith (1978) duration was 5 hours the fear assessment session duration of the present study lasted 30 minutes. In comparison, the fear assessment procedure of Mineka et al., (1981) and Miller et al., (1982) was 1 hour in duration. It is

believed the shorter duration used in the present experiment decreases possible variability in the dependent variables (i.e., ceiling effect reduced) as well as being a more economical use of time.

## Method

### Subjects

The subjects were 25 naive male New Zealand random bred Wistar rats from the Psychology Department's breeding colony. Their age range at the time of testing was 100-194 days with a mean age of 128 days. The maintenance schedule was identical to that of Experiment one.

### Apparatus

The apparatus was identical to that of experiment one except for the following modifications:

1. The two compartments were separated by a steel partition with an opening 9cm x 9cm (width x height) in its centre, flush with the grid floor. During RP treatment the aperture was closed by a stainless steel partition.
2. The stainless steel sheet over the grid floor of one compartment was removed giving both compartments identical grid floors.
3. The overhead illumination in the compartment in which shock was delivered, the fear conditioning (FC) compartment, remained on continuously throughout the experiment. Illumination changes in the other compartment, the safe compartment, was response dependent. That is, when the subject crossed from the safe to FC compartment, illumination

conditions in the safe compartment changed from flashing on-off to continuous illumination. When the subject was located in the FC compartment illumination was continuously on in both compartments. When the subject crossed from the FC to safe compartment, the illumination in the safe compartment changed from being continuously on to flashing on and off, (0.8 seconds off - 0.2 seconds on). Moving between compartments therefore affected the illumination conditions of the safe compartment.

The aversive unconditioned stimulus, shock, used during fear conditioning was delivered as for experiment one. The nominal setting for shock intensity was 0.5mA. Background white noise to mask extraneous sounds was approximately 65dB (A). Control apparatus was the same as for experiment one.

Procedure

Prior to the beginning of the experiment, subjects were randomly allocated to one of five groups (n = 5). An outline of the experimental design is presented in Table 2-2.

The experimental procedure was divided into four phases:

1. Habituation. On the first day of the experiment subjects were placed into the apparatus and allowed to freely move between both compartments for 30 minutes. During habituation, passive avoidance fear conditioning and the fear assessment phase. During passive avoidance extinction, three dependant variables were measured. They were:

(1) Total voluntary FC compartment time - this measure reflected the total cumulative time (sec.) during

TABLE 2-2

Outline of the experimental design.

| Treatment Groups | Days (sessions) | | | | | | |
|---|---|---|---|---|---|---|---|
| | 1(1) | 2(2) | 3 | 4(3) | 5(4) | 6(5) | 13(6) |
| Response Prevention Control (RPC) | Hab[a] | PA[b] | HC[d] | Ext[f] | Ext | Ext | Ext |
| Passive Avoidance Control (PAC) | Hab | AE[c] | HC | Ext | Ext | Ext | Ext |
| Response Prevention 1 hr massed (RP1) | Hab | PA | RP[e] | Ext | Ext | Ext | Ext |
| Response Prevention 2 hr massed (RP2) | Hab | PA | RP | Ext | Ext | Ext | Ext |
| Response Prevention 1 hr distributed (RPd) | Hab | PA | RP | Ext | Ext | Ext | Ext |

a Habituation to the apparatus

b Passive avoidance fear conditioning

c Exposure to apparatus cues without fear conditioning

d Home cage confinement

e Response prevention treatment in the FC compartment

f Passive avoidance extinction testing

the 30 minute sessions that the subject spent in the FC compartment.

(2) Total Number of approaches into the FC compartment - the number of times the subject voluntarily entered the FC compartment from the safe compartment.

(3) Average time spent in the FC compartment - this measure was defined as the time spent in the FC compartment per entry during the 30 minute sessions. That is, dividing the total time spent in the FC compartment by the number of entries into the FC compartment.

2. Fear Conditioning and Passive Avoidance Acquisition. Four groups received passive avoidance fear conditioning while one group, Group PAC, was placed into the apparatus and allowed access to both compartments as for habituation with no passive avoidance fear conditioning.

The subjects receiving passive avoidance fear conditioning (PAFC) were placed into the safe compartment of the apparatus and permitted to move between both compartments for the 30 minute session. During this session the delivery in the FC compartment of scrambled grid floor shock of 0.6 seconds duration was set up according to a V1-180 second schedule for subjects receiving PAFC. When delivery of the US was set-up, the V1-180 second timer stopped. For the US to be sampled the subject needed to move from the safe compartment to the FC compartment or be in the FC compartment. Depression of the FC compartment floor microswitch by the subject at this time initiated the delivery of the scrambled grid floor shock 4 seconds later. Shock offset began the V1-180 second timer. While the V1-180 second timer was running, movement in the FC compartment had no aversive

consequences, that is, the US was not set up for delivery. Typically, only 1 or 2 scrambled grid floor shocks were sampled by subjects. No subject totally avoided sampling at least one US delivery. This can be considered a passive avoidance procedure because subjects have to approach and enter the FC compartment to sample the US and because by staying in the safe compartment, i.e., remaining relatively inactive, the US can be avoided. However, it is also consistent to view this procedure as an intermittent positive punishment of crossings procedure. While I have termed the procedure, passive avoidance, the reader should be aware that this is not the only interpretation available to explain the procedure.

3. Response Prevention Treatment. On day three of the experiment, three groups received response prevention treatment in the experimental apparatus while the other two groups remained in their cages (Groups PAC and RPC). Subjects were placed into the FC compartment with access to the safe compartment prevented. Subjects received one of three RP treatments:

1. One hour massed RP treatment, Group RP1.
2. Two hours massed RP treatment, Group RP2.
3. One hour distributed RP treatment, Group RPd.

Distributed treatment comprised placing each subject into the FC compartment for 4-15 minute periods equally distributed over a 4 hour period.

4. Fear Assessment Measured In Extinction. All subjects were placed into the safe compartment of the apparatus with access to the FC compartment available. The shock source was disconnected for this phase of the experiment. Fear

extinction was measured during 30 minute sessions held on the 4th, 5th and 6th days of the experiment. The one-week follow-up assessment (day 13, cf. Table 2-2) was carried out to assess the time course of passive avoidance fear conditioning and the RP treatments.

Research Hypotheses

The experimental design generated the following research hypotheses:

1. During the first session of the experiment all subjects received identical treatment and therefore should show no difference in (i) the time spent in the FC compartment, (ii) the number of approaches into the FC compartment and (iii) the average time spent in the FC compartment.

2(a) During session 2 RPC, RP, RP2 and RPd subjects received fear conditioning while PAC subjects were exposed to the apparatus cues as in session one. If fear conditioning motivated subjects to avoid entry into the FC compartment then it is expected in comparison to PAC subjects those subjects receiving fear conditioning will (i) spend less cumulative time in the FC compartment, (ii) make fewer approaches into the FC compartment, and (iii) spend less average time in the FC compartment.

2(b) During session 2 if fear conditioning had the same effect on all subjects then RPC, RP, RP2, and RPd subjects should show no difference in (i) the cumulative time spent in the FC compartment, (ii) number of approaches into the FC compartment and (iii) the average time spent in the FC compartment.

3(a) If RP treatment reduced fear associated with the FC

compartment then RP subjects in comparison to RPC subjects (i) will spend more cumulative time in the FC compartment, (ii) make a greater number of approaches into the FC compartment and (iii) spend more average time in the FC compartment.

3(b) If RP treatment has completely eliminated fear of the FC compartment then RP subjects in comparison to PAC subjects should show no difference in (i) the cumulative time spent in the FC compartment, (ii) the number of approaches into the FC compartment and (iii) the average time spent in the FC compartment.

3(c) If the results of fear conditioning continues to motivate subjects from avoiding entry into the FC compartment then PAC subjects will in comparison to RPC subjects (i) spend more cumulative time in the FC compartment, (ii) make a greater number of approaches into thw FC compartment and (iii) spend more average time in the FC compartment.

3(d) If a difference exists between 1 hour massed RP treatment and 1 hour distributed RP treatment then it is expected RP1 subjects will in comparison to RPd subjects (i) spend either a greater or lesser amount of cumulative time in the FC compartment, (ii) make either more or less approaches into the FC compartment and (iii) spend either a greater or lesser amount of cumulative time in the FC compartment.

It should be noted hypotheses 3(a), 3(b), 3(c) and 3(d) also apply to sessions 3, 4, 5 and 6. The relationships outlined by these hypotheses could remain across these sessions.

4. If over the passive avoidance extinction sessions fear extinction takes place it is expected RPC subjects in

comparison to PAC subjects should show no difference in (i) the cumulative time spent in the FC compartment, (ii) the number of approaches into the FC compartment and (iii) the average time spent in the FC compartment.

## Results and Discussion

The raw scores yielded by the dependent variables were treated in the following way.

1. The total voluntarily FC compartment time was converted to proportion of session time spent in the FC compartment; by dividing FC compartment time by total session time. A score of 0.5 indicated equal distribution of times across the two compartments. A score of 1.0 indicated all the session time was spent in the FC compartment. A score of 0.0 indicated all the session time was spent in the safe compartment.
2. Number of approaches into the FC compartment was converted to a rate measure by dividing number of approaches by total session time (min.).
3. Average time in FC compartment per entry was used as measured. A preliminary split-plot ANOVA (Groups x Sessions) on this variable failed to yield either significant main effects for Groups or Sessions, or significant interaction. Since it was insensitive to experimental manipulations it was eliminated from further analyses.

A split-plot ANOVA (Kirk, 1968, p. 245) on the time spent in the FC compartment scores indicated that time varied across groups, ($F(4,20) = 5.43$, $p<.005$) varied over experimental sessions, ($F(5,20) = 7.98$, $p<.0001$) and this variation over experimental sessions was not constant for

each group yielding a significant groups by sessions interaction, ($F(20,100) = 3.77$, $p<.0001$).

A split-plot ANOVA on the approaches into the FC compartment per minute scores indicated approaches varied across groups, ($F(4,20) = 10.28$, $p<.0002$) varied across sessions, ($F(5,20) = 20.4$, $p<.0001$) and this variation over sessions was not constant for all groups yielding a significant groups by sessions interaction, ($F(20,100) = 3.12$, $p<.0002$).

Time Spent in the FC Compartment

Group means and standard deviations of the time measure across sessions are presented in Table 2-3, with the means being graphically represented in Figure 2-1.

There was no significant difference between groups in the time spent in the FC compartment on day one as indicated by the non significance of Tukey multiple comparison tests conducted between the groups on day one (refer Table 2-4). Overall, on day one, subjects spent 37% of their time in the FC compartment, indicating an initial slight bias towards the safe compartment.

The effect of fear conditioning can be examined by comparing day one and day two time scores. It can be seen from Figure 2-1 that all groups receiving fear conditioning reduced time spent in the FC compartment while the passive avoidance control group, PAC, had a slight increase in their time score. The reduction in the time measure was significant for groups RPC, RP2 and RPd, as indicated by multiple comparison Tukey tests, but not significant for the RP1 subjects (refer Table 2-5).

TABLE 2-3

Means and standard deviations of proportional time spent in the FC compartment for each group across sessions.

| Treatment Group | | Sessions 1 | 2 | 3 | 4 | 5 | 6 | Overall |
|---|---|---|---|---|---|---|---|---|
| RPC | M | 0.43 | 0.13 | 0.02 | 0.01 | 0.01 | 0.07 | 0.11 |
| | SD | 0.05 | 0.02 | 0.02 | 0.01 | 0.01 | 0.09 | 0.16 |
| PAC | M | 0.32 | 0.39 | 0.45 | 0.53 | 0.50 | 0.30 | 0.41 |
| | SD | 0.07 | 0.08 | 0.16 | 0.11 | 0.16 | 0.08 | 0.14 |
| RP1 | M | 0.39 | 0.20 | 0.21 | 0.24 | 0.20 | 0.25 | 0.25 |
| | SD | 0.04 | 0.15 | 0.13 | 0.07 | 0.09 | 0.11 | 0.12 |
| RP2 | M | 0.38 | 0.12 | 0.34 | 0.33 | 0.35 | 0.25 | 0.30 |
| | SD | 0.07 | 0.03 | 0.24 | 0.18 | 0.11 | 0.09 | 0.15 |
| RPd | M | 0.34 | 0.10 | 0.23 | 0.24 | 0.15 | 0.23 | 0.22 |
| | SD | 0.14 | 0.02 | 0.35 | 0.39 | 0.19 | 0.20 | 0.24 |
| Overall | M | 0.37 | 0.19 | 0.25 | 0.27 | 0.24 | 0.22 | |
| | SD | 0.08 | 0.13 | 0.24 | 0.25 | 0.21 | 0.14 | |

FIGURE 2-1 Proportional time spent in the FC compartment for each group across sessions.

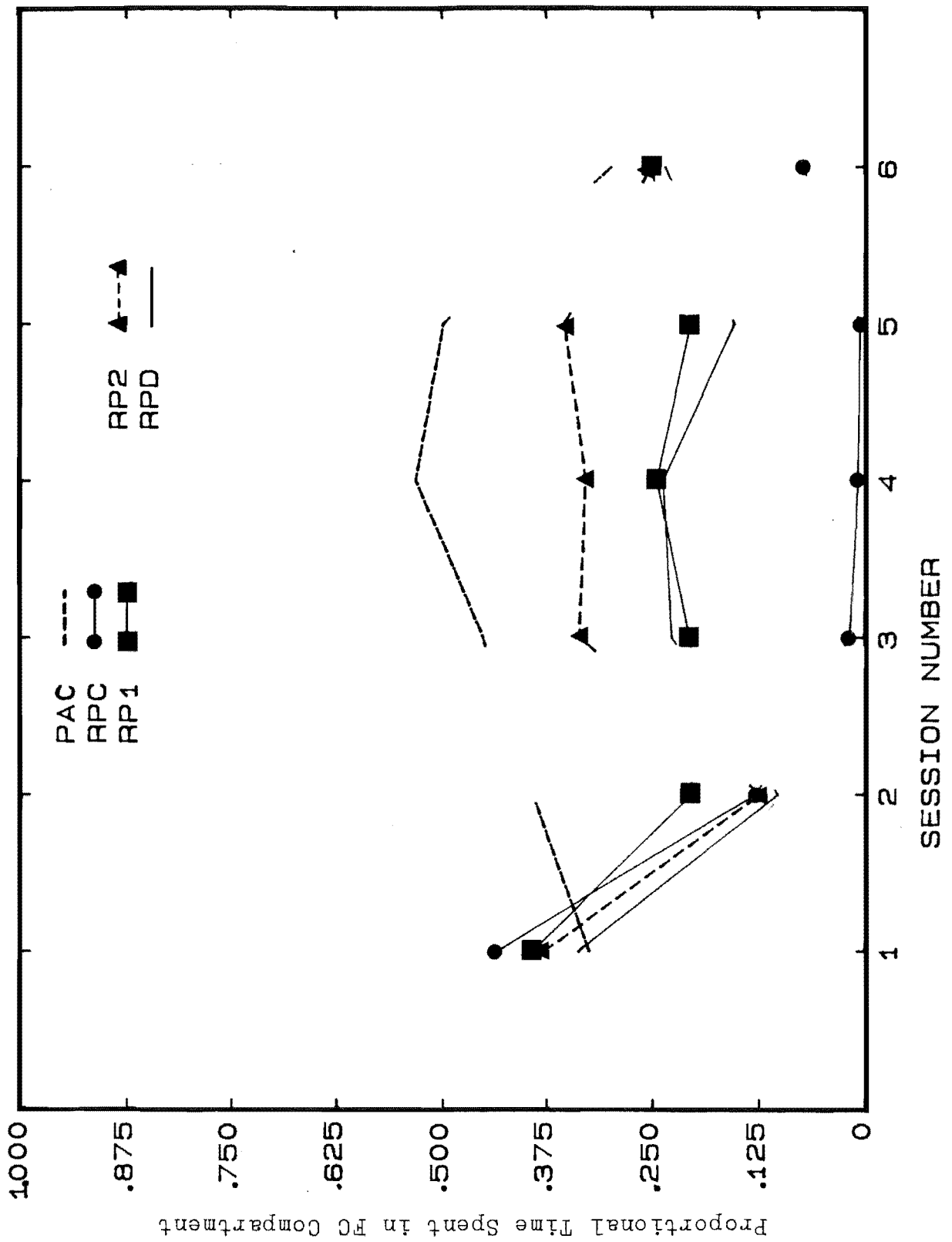


TABLE 2-4

Summary of the Tukey comparison tests for the Groups Factor at each level of the Sessions Factor : Proportional time data.

| Group Comparisons | Sessions | | | | | |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 |
| RPC-PAC | NS[a] | 3.96 | 6.58 | 7.92 | 7.48 | NS |
| RPC-RP1 | NS | NS | NS | 3.59 | 3.03 | NS |
| RPC-RP2 | NS | NS | 4.83 | 4.86 | 5.29 | NS |
| RPC-RPd | NS | NS | 3.20 | 3.47 | NS | NS |
| PAC-RP1 | NS | NS | 3.71 | 4.33 | 4.45 | NS |
| PAC-RP2 | NS | 4.03 | NS | 3.06 | NS | NS |
| PAC-RPd | NS | 4.38 | 3.38 | 4.45 | 5.23 | NS |
| RP1-RPd | NS | NS | NS | NS | NS | NS |
| RP1-RP2 | NS | NS | NS | NS | NS | NS |
| RPd-RP2 | NS | NS | NS | NS | 3.03 | NS |

a N.S. = non-significant result

$q'.05 = 2.889$

$q'.01 = 3.883$

TABLE 2-5

Summary of the Tukey comparison tests for the Sessions Factor as each level of the Groups Factor : Proportional time data.

| Session Comparisons | Groups | | | | |
|---|---|---|---|---|---|
| | RPC | PAC | RP1 | RP2 | RPd |
| 1-2 | 6.13 | NS | NS | 5.12 | 4.76 |
| 1-3 | 8.39 | NS | NS | NS | NS |
| 1-4 | 8.57 | 4.15 | NS | NS | NS |
| 1-5 | 8.63 | NS | NS | NS | NS |
| 1-6 | 7.28 | NS | NS | NS | NS |
| 2-3 | NS[a] | NS | NS | 4.27 | NS |
| 2-4 | NS | NS | NS | 4.14 | NS |
| 2-5 | NS | NS | NS | 4.64 | NS |
| 2-6 | NS | NS | NS | NS | NS |
| 3-4 | NS | NS | NS | NS | NS |
| 3-5 | NS | NS | NS | NS | NS |
| 3-6 | NS | NS | NS | NS | NS |
| 4-5 | NS | NS | NS | NS | NS |
| 4-6 | NS | 4.72 | NS | NS | NS |
| 5-6 | NS | NS | NS | NS | NS |

[a]NS = non-significant result

q.05 = 4.13

q.01 = 4.93

A closer examination of the individual time allocation scores of Group RP1 revealed one subject spent 47% of the session in the FC compartment although the overall group mean was only 20%. Examination of the data log for this subject indicated that a fault in the floor microswitches had been noted. This may have lead to a recording error of 47% proportional time in the FC compartment, a result misrepresenting the true situation. If this subject's score is removed from Group RP1, then the group mean becomes 14%, instead of 20%, which is very close to the other group means for groups receiving fear conditioning (refer Table 2-3).

The fear conditioning procedure produced an equivalent amount of avoidance of the FC compartment during session 2, since all Tukey multiple comparisons involving fear conditioning groups were non-significant (refer Table 2-4).

Extinction of passive avoidance responding was examined during sessions 3, 4 and 5. The passive avoidance control subjects, PAC, divided their time equally between compartments, spending 45%, 53% and 50% of the session in the FC compartment over the extinction sessions.

At the other extreme, response prevention controls, RPC, spent little time in the FC compartment over extinction sessions, 2%, 1% and 1%, respectively. The difference in performance between PAC and RPC subjects can be attributed to the effect of fear conditioning given to RPC subjects. This was a significant and reliable effect over the extinction sessions (refer Tables 2-4 and 2-5). It is clear that there was little attenuation in avoidance of the FC compartment over extinction sessions. Also, even though the RPC subjects experienced few CS-US pairings during session two, the

consequences were marked and durable over time.

Performance of PR1, RP2 and RPd subjects over the extinction sessions indicated that RP treatment significantly reduced the amount of fear associated with the FC compartment (refer Tables 2-4 and 2-5). Figure 2-1 shows RPC subjects time spent in the FC compartment decreased from session two (13%) to session three (2%) and remained at this level for the remaining extinction sessions. In contrast, the response prevention treated subjects' time spent in the FC compartment increased from session two (14%) to session three (26%) and remained at this level for the remaining extinction sessions. This consistency in the time measure over the extinction sessions 3, 4 and 5 is shown by the non significance of group comparisons for RPC, RP1, RP2 and RPd subjects presented in Table 2-5. Also, the time measure was significantly higher for response prevention subjects in comparison to RPC subjects (refer Table 2-4). Overall, the greatest amount of fear reduction resulted from the 2 hour massed RP treatment, followed by the 1 hour massed RP treatment and 1 hour distributed RP treatment which produced approximately the same amount of fear reduction. There was no significant difference between distributed or massed 1 hour RP treatment. Although all RP treatments increased the time spent in the FC compartment, it was still significantly below that of the PAC subjects, indicating some residual fear remained during the extinction sessions (refer Table 2-4).

One week following session five a follow-up session was conducted to assess the time course of fear conditioning and RP effects. During the follow-up session RPC subjects spent significantly less time in the FC compartment (7%) than

they did during the first session (43%). Clearly, conditioned fear of the FC compartment remained for these subjects. In comparison to RPC subjects, RP treated subjects allocated 25% of the session in the FC compartment. This indicates that there was no relapse by RP treated subjects upon follow-up, illustrating the durability over time of the RP treatments in reducing fear to the FC compartment.

Approaches per Minute into the FC Compartment

The group means and standard deviations of approaches per minute into the FC compartment are presented in Table 2-6 and graphically represented in Figure 2-2.

There was no significant difference between groups in approaches into the FC compartment during session one as indicated by the non significance of Tukey multiple comparison tests conducted between the groups on session one (refer Table 2-7).

During session two, all subjects except those in Group PAC received shocks, CS-US pairings, in the FC compartment, and reduced their entries into that compartment ($F(4,20) = 5.93$, $p<.003$). There were, however, greater differences between groups in the approach differences than in the time data. This might be a consequence of the way in which the shocks were scheduled. Not every entry was shocked, and up to 10 minutes of the 30 minute session would pass before a shock was received. This may account for the greater variability in performance during session two, as illustrated by the findings that only RPC and RPd subjects had fewer approaches into the FC compartment in comparison to PAC subjects.

TABLE 2-6

Means and standard deviations of approaches per minute into the FC compartment for each group across sessions.

| Treatment Group | | Sessions 1 | 2 | 3 | 4 | 5 | 6 | Overall |
|---|---|---|---|---|---|---|---|---|
| RPC | M | 0.95 | 0.40 | 0.14 | 0.09 | 0.05 | 0.23 | 0.31 |
| | SD | 0.32 | 0.08 | 0.19 | 0.15 | 0.06 | 0.24 | 0.36 |
| PAC | M | 0.78 | 0.71 | 0.72 | 0.60 | 0.58 | 0.82 | 0.70 |
| | SD | 0.10 | 0.17 | 0.17 | 0.20 | 0.20 | 0.34 | 0.21 |
| RP1 | M | 0.99 | 0.49 | 0.54 | 0.73 | 0.69 | 0.92 | 0.73 |
| | SD | 0.19 | 0.11 | 0.28 | 0.19 | 0.26 | 0.19 | 0.27 |
| RP2 | M | 0.84 | 0.63 | 0.59 | 0.56 | 0.61 | 0.67 | 0.65 |
| | SD | 0.09 | 0.21 | 0.21 | 0.28 | 0.15 | 0.41 | 0.24 |
| RPd | M | 0.61 | 0.35 | 0.17 | 0.23 | 0.31 | 0.44 | 0.35 |
| | SD | 0.21 | 0.08 | 0.09 | 0.12 | 0.29 | 0.20 | 0.22 |
| Overall | M | 0.83 | 0.52 | 0.43 | 0.44 | 0.45 | 0.62 | |
| | SD | 0.23 | 0.19 | 0.30 | 0.30 | 0.31 | 0.37 | |

FIGURE 2-2 Approaches per minute into the FC compartment for each group across sessions.

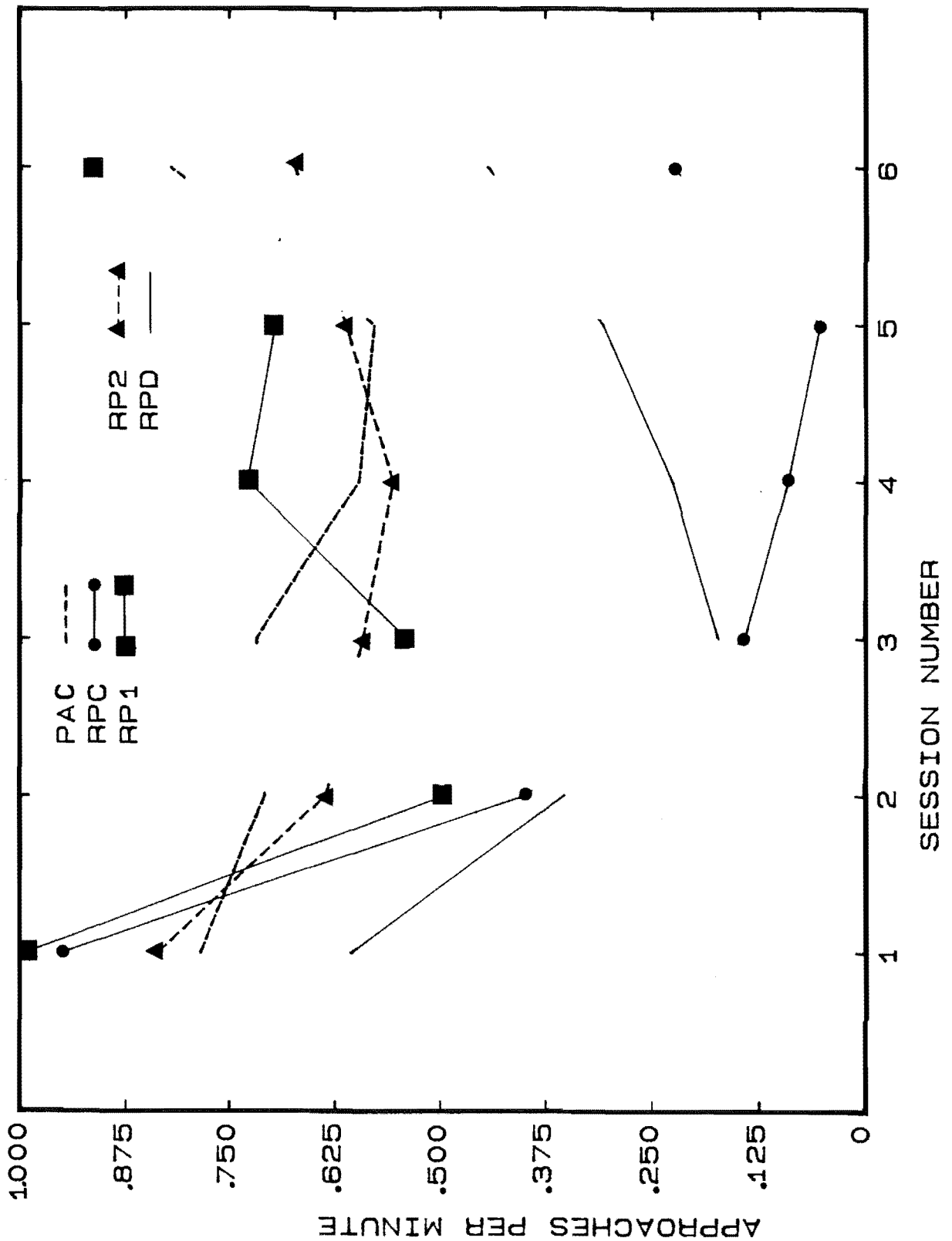


TABLE 2-7

Summary of the Tukey comparison tests for the Groups Factor at each level of the Sessions Factor : Approaches into the FC compartment data.

| Group Comparisons | Sessions | | | | | |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 |
| RPC-PAC | NS[a] | 3.28 | 6.13 | 5.38 | 5.60 | 6.33 |
| RPC-RP1 | NS | NS | 4.24 | 6.79 | 6.83 | 7.35 |
| RPC-RP2 | NS | NS | 4.79 | 4.96 | 5.96 | 4.74 |
| RPC-RPd | NS | NS | NS | NS | NS | NS |
| PAC-RP1 | NS | NS | NS | NS | NS | NS |
| PAC-RP2 | NS | NS | NS | NS | NS | NS |
| PAC-RPd | NS | 3.78 | 5.79 | 3.92 | 2.89 | 4.00 |
| RP1-RPd | NS | NS | 3.90 | 5.33 | 4.13 | 5.02 |
| RP1-RP2 | NS | NS | NS | NS | NS | NS |
| RPd-RP2 | NS | 3.01 | 4.45 | 3.50 | 3.25 | NS |

a NS = non-significant result

$q'.05 = 2.88$

$q'.01 = 3.86$

All groups receiving shocks during session two should show no differences in approaches into the FC compartment. This was found as no comparisons reached statistical significance (refer Table 2-7). However, fear conditioning clearly reduced to a greater degree approaches into the FC compartment by RPd subjects in comparison to other groups (see Figure 2-2).

Across extinction sessions 3, 4 and 5, PAC subjects reduced the number of approaches into the FC compartment from 0.72 to 0.58 per minute, although this reduction failed to reach statistical significance as illustrated in Table 2-8. Approaches into the FC compartment for RPC subjects decreased from 0.14 to 0.09 per minute across extinction sessions. This indicated that fear of the FC compartment was not attenuated by the mere passage of time, but rather, subjects were increasingly avoiding entering the FC compartment. PAC subjects entered the FC compartment during each extinction session on significantly more occassions than RPC subjects (refer Table 2-7). This corroborates the time measure findings as previously reported.

Performance of the RP1 and RP2 subjects over the extinction sessions indicated that massed RP treatments significantly reduced the amount of fear associated with the FC compartment (refer Table 2-7). Distributed RP treatment failed to increase the number of entries into the FC compartment in comparison to RPC subjects across extinction sessions. Figure 2-2 shows RPC subjects' approaches into the FC compartment decreased from session two (0.40) to session three (0.14) and further decreased over the remaining extinction sessions. In contrast, the massed RP treated

TABLE 2-8

Summary of the Tukey comparison tests for the Sessions Factor at each level of the Groups Factor : Approaches into the FC compartment data.

| Session Comparisons | Groups | | | | |
|---|---|---|---|---|---|
| | RPC | PAC | RP1 | RP2 | RPd |
| 1-2 | 7.18 | NS[a] | 6.48 | NS | NS |
| 1-3 | 10.52 | NS | 5.88 | NS | 5.61 |
| 1-4 | 11.18 | NS | NS | NS | 4.91 |
| 1-5 | 11.67 | NS | NS | NS | NS |
| 1-6 | 9.43 | NS | NS | NS | NS |
| 2-3 | NS | NS | NS | NS | NS |
| 2-4 | NS | NS | NS | NS | NS |
| 2-5 | 4.49 | NS | NS | NS | NS |
| 2-6 | NS | NS | 5.48 | NS | NS |
| 3-4 | NS | NS | NS | NS | NS |
| 3-5 | NS | NS | NS | NS | NS |
| 3-6 | NS | NS | 4.88 | NS | NS |
| 4-5 | NS | NS | NS | NS | NS |
| 4-6 | NS | NS | NS | NS | NS |
| 5-6 | NS | NS | NS | NS | NS |

[a]NS = non-significant result

q.05 = 4.13

q.01 = 4.93

subjects' approaches into the FC compartment remained the same from session two to session three (0.56) and increased to 0.65 approaches per minute during session five. This increase of approaches into the FC compartment by massed RP treated subjects across extinction sessions was comparable to PAC subjects, indicating massed RP treatment had eliminated fear of the FC compartment assessed by the approaches measure (refer Tables 2-7 and 2-8). In contrast, a significant difference between RPd and PAC subjects on the approaches measure remained across extinction sessions, indicating some residual fear of the FC compartment by RPd subjects.

During the follow-up session RPC subjects entered the FC compartment on significantly less occassions than they did during session one (refer Table 2-8). Clearly, RPC subjects still avoided entering the FC compartment indicating fear of this compartment still remained. In comparison to RPC, massed RP treated subjects entered the FC compartment on average 0.79 times per minute during the follow-up session. The massed RP treatment effect was persistent over time and completely eliminated conditioned fear of the FC compartment when assessed by the approaches measure.

Both the approaches and time measures indicated fear conditioning, as conducted in this experiment, had a profound effect on behaviour, an effect that was durable and persistent for a number of days. Both measures were sensitive to RP effects on conditioned fear reduction, with the time measure having greater consistency and less variability than the approaches measure.

## Correlations Between the Conditioned Fear Assessment Measures

In order to assess if any relationship exists between the time and approaches measures, Pearson product-moment correlation coefficients were calculated across all subjects for each session. A summary of the obtained correlation coefficients is given in Table 2-9.

To further determine whether any relationship across subjects represents a between-groups relationship, correlation coefficients were computed between these two fear assessment measures using group means. The obtained correlation coefficients are presented in Table 2-10.

It is clear from examination of the correlation coefficients that a positive relationship exists between both fear assessment measures. Given this relationship it was considered appropriate to further examine the relationships established with the univariate analyses just presented by conducting a two-way Groups x Sessions multivariate analysis of variance, MANOVA, with repeated measures on the sessions factor.

### Multivariate Analysis of Variance

BMDP4V (Dixon, 1981) MANOVA computer programme was used for all analyses.

With the use of Wilk's Lamba likelihood ratio criterion, the combined dependent variables, approaches into the FC compartment and time spent in the FC compartment, were significantly affected by both group allocation, ($F(8,38) = 5.91$, $p<.001$), and sessions, ($F(10,198) = 11.56$; $p<.001$), and by their interaction, ($F(40,198) = 2.97$, $p<.001$). The results

TABLE 2-9

Pearson product-moment correlation coefficients between the dependent variables for all subjects.

| | Sessions | | | | | |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 |
| Correlation coefficient (r) | 0.4835 | 0.3809 | 0.5004 | 0.3868 | 0.5638 | 0.5421 |
| Probability level (p) | 0.007 | 0.030 | 0.005 | 0.028 | 0.002 | 0.003 |

n = 25, df = 23, two-tailed test.

TABLE 2-10

Pearson product-moment correlation coefficients between the dependent variables for group means.

| | Sessions | | | | | |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 |
| Correlation coefficient (r) | 0.7661 | 0.7205 | 0.8320 | 0.6872 | 0.7408 | 0.8434 |
| Significance Probability level (p) | 0.065 | 0.085 | 0.040 | 0.100 | 0.076 | 0.036 |

n = 5, df = 2, two-tailed test.

reflected a high degree of association between group allocation and the combined dependent variables, $\eta^2 = 0.80$ That is, 80% of the variance in the linear combination of the two dependent variable scores is accounted for by assignment to experimental groups. There is only a moderate degree of association between sessions and the dependent variables, $\eta^2 = 0.60$.

Given that a significant difference between treatment groups exists it is of interest to ascertain which of the two dependent variables is affected by independent variable manipulation, and which, if any, remains unaffected. Analyses of covariance were conducted to examine this possibility. In the first analysis of covariance the approaches measure was the dependent variable with the time scores acting as a covariate. Approaches into the FC compartment varied across group allocation, ($F(4,19) = 6.72$, $p<.002$), across sessions, ($F(5,99) = 15.7$, $p<.001$), and by their interaction, ($F(20,99) = 2.3$, $p<.01$). In the second analysis of covariance, the approach scores acted as a covariate. Time spent in the FC compartment varied across group allocation, ($F(4,19) = 3.07$, $p<.05$), across sessions, ($F(5,99) = 4.78$, $p<.001$) and by their interaction, ($F(20,99) = 2.86$, $p<.001$). Clearly, both fear assessment measures made unique contributions to the composite dependent variable used in the MANOVA analysis.

To ascertain the relative contribution of the fear assessment measures to the multivariate discrimination, a stepwise discriminant function analysis was performed using the time and approach measures as predictors of membership in the five treatment groups. A summary of the results is

presented in Table 2-11. The analysis yielded two discriminant functions, with a combined Chi Squared of ($X^2(8) = 83.56$, $p<.001$). The two discriminant functions accounted for 79.87% and 20.13%, respectively, of the between-group variability, (see Table 2-11(a)). Thus, the groups differed in at least two significant ways. The canonical discriminant functions evaluated at group means (group centroids) are plotted in Figure 2-3. As can be seen, the first discriminant function maximally separates PAC, RP1 and RP2 groups from RPC and RPd groups. In other words, with regard to the first discriminant function PAC, RP1 and RP2 groups are indistinguishable from each other, while the RPd group most closely resembles the RPC group. The second discriminant function maximally separates RPd and PAC groups from the RP1 group, with RP2 and RPC groups falling in between the other groups. Here, RPd and PAC groups are indistinguishable from each other, as are RPC and RP2 groups, with the RP1 group on its own.

The loading matrix (pooled within-groups correlations) between the measures and discriminant functions, see Table 2-11(b), indicates that the first discriminant function is correlated most highly with the approaches measure ($r = 0.892$), although it is also correlated highly with the time measure ($r = 0.743$); while the second discriminant function is primarily loaded with the time measure ($r = 0.669$), with only moderate correlation with the approaches measure ($r = -0.452$. Relating these findings to the plot of group centroids, Figure 2-3, it suggests that the maximum separation among the groups on the first discriminant function is primarily based on the approaches measure scores although not entirely,

TABLE 2-11

(a) Canonical discriminant functions - Summary Table

| Function | Eigenvalue | % of Variance | Canonical Correlation | After Function | Wilkes Lambda | $X^2$ | df | Significance |
|---|---|---|---|---|---|---|---|---|
| 1 | 0.557 | 79.87 | 0.598 | 0 | 0.563 | 83.558 | 8 | 0.000 |
| 2 | 0.140 | 20.13 | 0.351 | 1 | 0.877 | 19.122 | 3 | 0.000 |

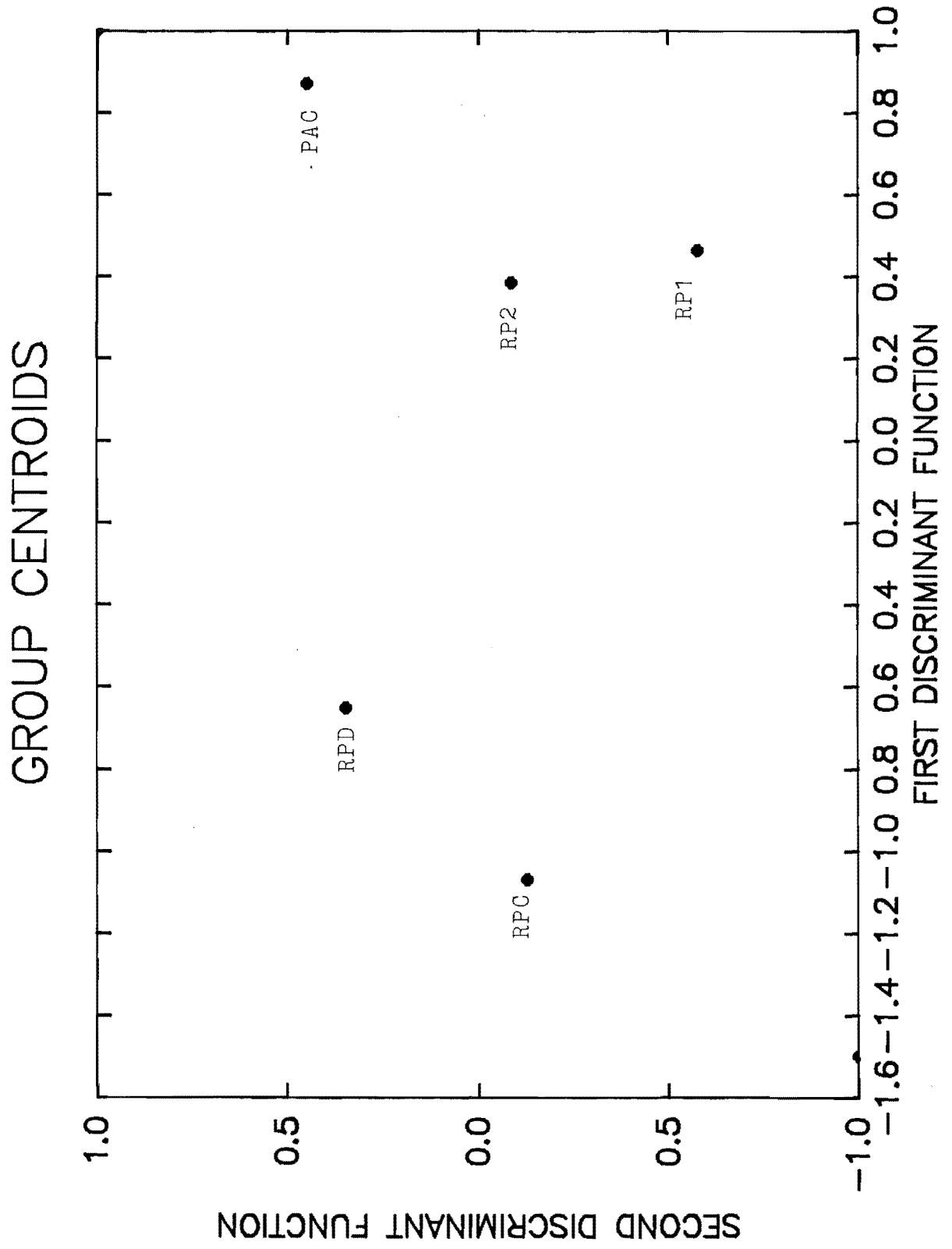
(b) Loading Matrix between predictor variables and discriminant functions.

| Measure | Function 1 | Function 2 |
|---|---|---|
| Time | 0.743 | 0.669 |
| Approach | 0.892 | -0.452 |

(c) Standardised Canonical Discriminant Function Coefficients

| Measure | Function 1 | Function 2 |
|---|---|---|
| Time | 0.485 | 0.956 |
| Approach | 0.717 | -0.797 |

FIGURE 2-3 Group centroids in the discriminant space formed by the first and second discriminant functions.



with the greatest difference obtained between RPC and PAC groups, as expected. Examining the second discriminant function, the RPd and PAC groups are distinguished from the RPC and RP2 groups which are in turn distinguished from the RP1 group, primarily on the basis of the time measure. Yet at the same time, RPd was indistinguishable from PAC, and RPC was indistinguishable from PR2 on the second discriminant function. However, the first discriminant function whose effect is primarily due to the approaches measure, with a lesser contribution from the time measure, did separate the massed RP treatments and passive avoidance control subjects from the RPC and RPd groups. On the basis of the foregoing information it seems that the approaches measure most effectively separated the RP treatment groups from the non RP treatment groups and extrapolating from these findings, was the most sensitive fear assessment measure. This conclusion was confirmed by the result of the stepping procedure used to enter the measures into the discriminant function analysis. The analysis employed maximum changes in Rao's V (largest increase in distance between groups) as the criteria for including measures into the analysis. The measure or variable with the greatest discriminating power is entered first, followed by the variable with the next highest discriminating power, and so on. A variable is retained in the analysis only if it significantly added discriminating power to the discriminant function equation. The approaches measure was entered first into the discriminant analysis, followed by the time measure. Both measures produced significant changes in Rao's V when included in the analysis, for the approaches measure (change in Rao's V

= 68.45, $p<.001$) and for the time measure (change in Rao's V = 32.71, $p<.001$). Thus, both measures made significant contributions to the discriminant function analysis. This finding confirms the results of the analyses of covariance previously reported. This result also indicated that the approaches measure had a higher discriminating power relative to the time measure, that is, the most sensitive fear assessment measure.

The results of this experiment further confirm the usefulness of the approach methodology technique in RP studies (Corriveau & Smith, 1978). Response prevention treatment produced substantial fear reduction as assessed by two related but different indices of conditioned fear. A third index, average time spent in the FC dompartment per entry, proved insensitive in discriminating the different RP treatments from control conditions.

The present experiment is one of the few to use a protracted RP treatment duration. By comparison, the majority of earlier RP studies used a RP duration of 5 minutes (Baum, 1970; Smith, Dickson & Sheppard, 1973). Mineka and Gino, (1979a) and Mineka et al., (1981) used a RP duration of 40 minutes; Mineka and Gino (1979b) and Corriveau, Contildes and Smith (1978) used a RP duration of 60 minutes and the present experiment is the only RP treatment study to use a RP duration of 2 hours.

In earlier studies RP treatment effects were confounded with the passage of time. Response prevention was administered between avoidance acquisition and extinction testing phases of the experimental procedure, whereas no RP treated controls were given extinction testing immediately

upon the completion of avoidance acquisition. The present experiment equated the passage of time of across experimental groups.

The present experiment found massed RP treatment to be superior to distributed RP treatment in facilitating conditioned fear and avoidance extinction. A number of explanations could account for this finding.

Massed extinction trials, by shortening the intertrial interval, increase the rate of extinction for both Pavlovian and Instrumental conditioning procedures (Pavlov, 1927; Mackintosh, 1974). If non-reinforced exposure to the CS, RP treatment, can be regarded as analogous to a series of extinction trials, then massing CS exposure should result in faster fear and avoidance extinction. Within the context of the RP paradigm a massed trial extinction procedure has been found superior in facilitating conditioned fear and avoidance extinction in comparison to a standard RP treatment (Baum & Oler, 1968) and this superiority remained when both procedures were equated for non-reinforced CS exposure duration (Blampied & Samuels, 1983).

The conservation of anxiety principle (Solomon & Wynne, 1954; Levis, 1979) proposed conditioned fear is conserved to parts of the CS-UCS interval temporally distal from CS onset. Thus, the shorter the non-reinforced CS exposure, the more conditioned fear is conserved and the less conditioned fear is reduced. A massed procedure should be superior in facilitating conditioned fear and avoidance extinction in comparison to a distributed procedure with multiple short duration non-reinforced CS exposures. Contrary to the predictions of the conservation of anxiety principle,

a number of researchers have reported a series of short duration non-reinforced CS exposures was more effective in hastening avoidance extinction than one single massed RP trial (Baum & Myran, 1971; Berman & Katzev, 1972; Franchina, Agee & Hauser, 1974). This result supports Rescorla's contingency theory of Pavlovian conditioning which maintains each non-reinforced CS exposure reduces the CS-UCS contingency (Rescorla, 1967). Extinction of conditioned fear and avoidance responding is a function of the number of non-reinforced CS exposures with total non-reinforced CS exposure having little effect.

Shipley, Mock and Levis (1971) stated the number and duration of non-reinforced CS exposures had little effect on conditioned fear and avoidance extinction. Rather, the cumulative total non-reinforced CS exposure duration determines the rate of conditioned fear and avoidance extinction. This explanation predicts no difference between massed vs distributed RP treatments provided the cumulative total non-reinforced CS exposure duration is the same for both procedures. This explanation predicts the RP1 and RPd result of the present study while also illustrating the potency of total non-reinforced CS exposure duration in determining conditioned fear and avoidance extinction. It is known that total non-reinforced CS exposure duration is an important variable in the efficacy of RP treatment as a conditioned fear reduction procedure (Mineka, 1979; Mineka & Gino, 1979b). It would seem this represents the most parsimonious explanation of the massed vs distributed RP treatment result of the present experiment. A number of other researchers have also reported no differences between massed vs

distributed RP treatment (Bankart & Elliott, 1974; Schiff, Smith & Prochaska, 1972; Shearman, 1970; Shipley, 1974).

Recently, the question has been asked; is exposure a necessary (and sufficient) condition for fear reduction? (DeSilva & Rachman, 1981, 1983; Boyd & Levis, 1983; Gelder, 1982). First some definitions of what is meant by necessary and sufficient conditions. A condition is considered necessary when its removal, while all other conditions remain constant, results in the absence of the phenomenon under investigation (Stebbing, 1961). Thus, to determine a condition is necessary one experimentally excludes the necessary condition and contrasts the results with a control treatment procedure where the necessary condition is included along with all other conditions.

According to Stebbing (1961) a condition is regarded as sufficient when its presence results in the induction of the phenomenon. Thus, to determine if a condition is sufficient one would experimentally include the 'sufficient' condition in isolation from other conditions in the experimental treatment group and contrast the results with a control treatment procedure which excludes the sufficient condition.

De Silva and Rachman (1981) argued while non-reinforced CS exposure maybe a sufficient condition for fear-reduction it is not a necessary condition. De Silva and Rachman provided examples of fear reduction in the absence of non-reinforced CS exposure to support their position:

> (a) the common clinical and experimental observation that imparting information about the harmlessness of the stimulus can lead to a reduction of fear; (b) suggestive evidence that cognitive therapy produces fear-reduction; (c)

> spontaneous remissions of neurotic, including anxiety, reactions in a proportion of patients; (d) improvement observed after administration of placebos ... (p.230).

Boyd and Levis (1983) have replied to De Silva and Rachman's comments by arguing the Pavlovian principle of experimental extinction with non-reinforced CS exposure producing conditioned fear extinction should not be readily abandoned as advocated by De Silva and Rachman. Boyd and Levis (1983) incorporated examples of fear reduction in the absence of non-reinforced CS exposure used by De Silva and Rachman (1981) into a number of theories stressing the importance of non-reinforced CS exposure in fear reduction (Eysenck, 1968, 1979; Levis, 1979; Levis & Hare, 1977; Stampfl & Levis, 1967, 1969, 1976).

Boyd and Levis (1983) called for a more careful analysis of the condition fear-avoidance extinction process. They focused their analysis on fear reduction treatments based on non-reinforced CS exposure while De Silva and Rachman (1981) argues for an analysis of the conditions under which conditioned fear is reduced in the absence of non-reinforced CS exposure. Agreement has been forged regarding non-reinforced CS exposure as being a sufficient condition for conditioned fear and avoidance reduction, but the question of whether non-reinforced CS exposure is a necessary condition for fear reduction remains hotly contested between the respective protagonists.

Overall, the results of the present experiment are consistent with the modified two-factor theory of fear-avoidance extinction. According to this theory, increasing

RP treatment duration should facilitate fear-avoidance extinction through the process of Pavlovian fear extinction occurring during non-reinforced exposure to the CS complex. Subjects receiving 2 hours of RP treatment spent more time in the FC compartment and made more approaches into the FC compartment during passive avoidance extinction testing than subjects which received 1 hour RP treatment or subjects that acted as RP controls. The absence of residual fear for the RP2 subjects indicated RP2 treatment had completely eliminated conditioned fear to the FC compartment and contextual cues while residual fear was still present after 1 hour RP treatment.

The results of the present experiment are also consistent with the effective reinforcement hypothesis (McAllister, McAllister & Dieter, 1976; McAllister, McAllister, Dieter & James, 1979) because the longer duration of RP treatment should bring about greater extinction of contextual fear as well as conditioned fear to the CS. It is known fear is conditioned to both a nominal CS complex and contextual cues during avoidance acquisition (Bouton, 1982; Bouton & Bolles, 1979a, 1979b; Bouton & King, 1983; McAllister et al., 1983).

Competing response theory (Linton, Riccio, Rohrbaugh & Page, 1970; Page, 1955) proposes conditioned fear is not reduced by RP treatment, but rather the avoidance response is replaced by a competing response learned during non-reinforced exposure to the CS. This competing response, e.g., freezing or crouching, results in the extinction of the learned avoidance response but not in the extinction of conditioned fear. Much of the research supporting competing response theory rests on the result of residual fear being

greater following RP treatment (Page, 1955; Coulter, Riccio & Page, 1969). The present experiment failed to show a residual fear effect for subjects receiving 2 hours of RP treatment and clearly fails to support this theory. In the present experiment, a change in illumination conditions in the presence of the subject in the FC compartment acted as the nominal CS. Recently, Sigmundi, Bouton and Bolles (1980) and Sigmundi and Bolles (1983) reported less conditioned freezing (an incompatible or competing response) to a light CS in comparison to a noise to generate the CS. Given this result, the present experimental conditions were less likely to generate competing response patterns during exposure to the light CS in the FC compartment. Also, it must be remembered that learning of a freezing response during RP when approach latency procedures are used functions to inhibit the subject from approaching the FC compartment from the safe compartment and voluntarily remaining in the FC compartment. The results of the present experiment fail to support this expectation. In essence, it is extremely difficult to present a competing response interpretation when using approach methodology dependent variables.

A theory very similar to the competing response theory is the species specific defense reactions (SSDR) proposed to account for RP treatment effects (Bersh & Keltz, 1971; Crawford, 1977). While competing response theory proposes responses such as freezing are <u>learned</u> during RP treatment SSDR theory proposes responses such as freezing are <u>innate</u>, part of the subjects defensive response behavioural repertoire. The RP treatment increases the probability of freezing becoming the dominant response by preventing the

flight SSDR. Irrespective of the source of the competing response pattern, learned or innate SSDR, the points raised considering the adequacy of the competing response theory to the results of the present experiment are also applicable to the SSDR theory.

Crawford (1977) stated:

> ... the SSDR hypothesis predicts that when measures of fear other than avoidance responding are given during or after response prevention, those measures will reflect the high level of fear present in the situation. (p. 51).

In support of this prediction she mentioned the result of RP treated subjects being more reluctant to enter a previously aversive chamber for food than regular extinction treated subjects (Coulter, et al., 1969; Linton, et al., 1970). While in the present experiment food was absent from the FC compartment, our RP treatment subjects made significantly more approaches into the FC compartment in comparison to controls and failed to evidence the high levels of fear predicted by SSDR theory.

Baum's (1970) relaxation theory of RP treatment and Seligman and Johnston's cognitive theory both explain the results of the present experiment. Baum maintained during RP treatment subjects learn to relax and relaxation responses replace and inhibit avoidance responses to the CS. These relaxation responses are believed to be emitted from 160 seconds after the beginning of the RP treatment (Baum, 1969b). Given the present experiment used RP durations of 60 and 120 minutes it is clear relaxational responses could have been learned. The longer the RP treatment the more likely these responses would have been emitted and the effective RP

treatment is in facilitating fear avoidance extinction. The major problem with this analysis is that given the RP duration is greater than 160 seconds and a significant RP effect is obtained, then a relaxational analysis could explain the RP effect. The relaxation analysis becomes very difficult to disprove unless RP treatment of greater than 160 seconds has no effect on reducing conditioned fear and avoidance responding. Another weakness of this theory is its failure to explain conditioned fear, no relaxational responses following RP treatment of greater than 160 seconds duration, the residual fear effect, which has been obtained by some researchers (Coulter et al., 1969; Page, 1955).

Seligman and Johnston's (1973) cognitive theory maintains during avoidance acquisition the subject learns two expectancies, firstly, that responding leads to shock (passive avoidance paradigm), and secondly, that not responding leads to something other than delivery of the unconditioned aversive stimulus. The function of response prevention and also regular extinction trials is to disconfirm and weaken the above expectancies and to strengthen the expectancy that responding does not lead to UCS delivery (passive avoidance paradigm). Again, if a RP treatment effect is obtained then it can be explained in terms of cognitive theory. The difficulty is to independently assess changes in cognitive expectancies, in other words, the theory goes outside the realms of testability (Mineka, 1979).

Because of the similarities between conditioned avoidance responses in animals and fear-anxiety motivated neurotic reactions in humans (Baum & Poser, 1971; Leitenberg, 1976; Levis, 1981) researchers have been interested in

investigating treatment procedures for eliminating conditioned fear, anxiety and avoidance responses. Several researchers have shown passive avoidance approach assessment techniques, such as latency to approach a feared CS complex and the total time a subject voluntarily exposes himself to the CS complex during fear assessment testing, provide sensitive measures of conditioned fear and anxiety (Corriveau & Smith, 1978; Mineka et al., 1981; Miller et al., 1982; the present experiment).

# EXPERIMENT THREE

## Facilitated Extinction of Conditioned Avoidance Responding: The Effects of Nonfearful Subjects, Propranolol, Atenolol and Diazepam as Adjuncts to Response Prevention Treatment.

### Introduction

Experiment two demonstrated the facilitative effects of RP treatment on hastening passive avoidance extinction using the approach method of Corriveau and Smith (1978). A number of procedures have been used in conjunction with RP treatment to increase the ability of RP to hasten avoidance-fear extinction. Two procedures are further examined in this experiment: the social facilitation effect (Baum 1969c; Corriveau, Contildes & Smith, 1978; Reynierse, Klomp & Bach, 1974) and drug assisted RP treatment (Baum, 1973; Christy & Reid, 1975; Cooper, et al., 1974; Kamano, 1968, 1972; Taub, et al., 1977). Two drug classes were chosen: benzodiazepine and beta-adrenergic blockers.

Anxiolytics have been defined as a class of psychotropic compounds with distinctive biological-behavioural effects (Fielding & Lal, 1979) which have been used in the treatment of anxiety associated disorders (Leavitt, 1974). The first chemical agent used for the treatment of such symptoms was meprobamate (Hill & Tedeschi, 1971).

However, the most potent and widely used anxiolytics are the benzodiazepines: chlordiazepoxide, diazepam, oxazepam and lorazepam, to name a few.

Anxiolytics are now in wide use (Tallman, Paul, Skolnick

& Gallager, 1980) and are also prescribed as muscle relaxants, anticonvulsants or hypnotics (Goodman & Gilman, 1975). Although benzodiazepines are among the safest therapeutic drugs, they can produce a number of adverse reactions (Dukes, 1980), although recent benzodiazepines appear free from adverse side-effects (Gschwend, 1979).

Because of the proliferation of new compounds which may have potential as anxiolytic drugs, there is a need for efficient pre-clinical screening tests of their effects. These pre-clinical screening tests typically employ animals, and serve the purpose of providing a means of assessing the safety and efficacy of any new compound. They are preferred to human screening tests because of the dangers and ethical difficulties involved in studying untested compounds on humans. With the elucidation of mechanisms of drug action, new drugs can be synthesized which are more selective in action, safer and more efficient (Lippa, Nash & Greenblatt, 1979).

If valid conclusions about drug effects on humans are to be made, then animal screening tests should meet a number of criteria. These are, 1. screening tests should be sensitive in a dose-dependent fashion, 2. the relative potency of known psychotropic drugs in animal screening tests should compare favourably to their relative potency in humans and 3. a screening test should be sensitive to one class of compounds but insensitive to other classes (Glick, 1976).

During the past two decades a number of animal screening tests have been developed to identify compounds that have potential anxiolytic action. The tests can be divided into those which use untrained responses and those which use

trained responses. Because screening tests on untrained responses such as aggressive, consumatory and exploratory behaviours are brief and require little or no training of the animal before testing they have found widespread use by psycho-pharmacologists. Trained subjects are used to measure potential anxiolytic effects on conditioned avoidance responding, conditioned emotional responding and punished operant behaviour.

One of the factors that lead to the use of benzodiazepines in humans was the finding that they appeared to inhibit animal aggressive behaviour (Haefeley, 1978; Valzell, 1973). This anti-aggressive effect of anxiolytics has been demonstrated through reduction in isolation induced mouse fighting (Malick, 1978; Valzelli, 1973; Yen, Stanger & Millman, 1959), foot-shock induced mouse fighting (Tedeschi, et al., 1959; Sternbach, et al., 1964; Valzelli, 1967, Zbinden & Randall, 1967), defensive aggression in cats (Hoffmeister & Wottke, 1969), and monkey aggressive behaviour (Heise & Boff, 1961; Scheckel & Boff, 1967).

While this effect is demonstrated across a number of species, some anxiolytic agents have failed to produce the effect (Randall, et al., 1965; Scheckel & Boff, 1967) and non-anxiolytic agents have produced it (Hoffmeister & Wottke, 1969; Valzelli, 1967). This lack of selectivity has lead some authors to propose the anti-agressive effect as being a unreliable indicator of anxiolytic efficacy (Glick, 1976; Lippa, et al., 1979).

One effect of benzodiazepines is to increase consumatory behaviour (Boissier, Simon & Soubrie, 1976; Hanson & Stone, 1964). While the relative potency of some anxiolytics (e.g.

chlordiazepoxide, diazepam, oxazepam and nitrazepam) measured using the consumatory screening test is equivalent to their potency in the treatment of human anxiety, two earlier developed anxiolytics, meprobamate and pentobarbital, fail to increase consumatory behaviour (Poschel, 1971).

Research has also shown that anxiolytics increase exploratory behaviour in a novel environment (Britton & Britton, 1980, 1981; Christmas & Maxwell, 1970; Hughes, 1972; Marriott & Smith, 1972; Marriott & Spencer, 1965; Crawley, 1981). In short, screening tests using untrained behaviour are not wholly adequate.

One procedure involving trained responses is the operant punishment procedure which includes rewarding and punishing an operant response emitted by the subject (Geller & Seifter, 1960; Geller, et al., 1962). Typically, anxiolytics have been found to disinhibit behaviour suppressed by the punishment component of the conflict procedure (Cook & Davidson, 1973; Lippa, et al., 1978; Margules & Stein, 1968; Vogel, et al., 1971). The anticonflict potency of anxiolytics is equivalent to their anti-anxiety potency with humans (Greenblatt & Shader, 1974). Both food (Geller & Seifter, 1960; Cook & Davidson, 1973) and water (Miczek & Lau, 1975; Kilts, et al., 1981) have been used to provide positive reinforcement in the conflict test. The anticonflict effect of anxiolytics has been demonstrated across a wide variety of species including rats (Cook & Sepinwall, 1975a, b, c), cats (Masserman, 1957, 1959), pigs (Dantzer & Roca, 1974), goldfish (Kelleher & Morse, 1964, 1968), squirrel monkeys (Hanson, et al., 1967; Glowa & Barrett, 1976) and humans (Beer & Migler, 1975). Punished operant procedures have

yeilded consistent results where other screening tests have not.

Another procedure used in the detection of potential anxiolytic agents is the conditioned emotional response (CER) test, or as it is sometimes termed the Estes-Skinner procedure, named after the researchers who designed the procedure (Estes & Skinner, 1941). The procedure involves two concurrently presented phases. First the operant conditioning phase involves operant responding (e.g., barpressing or licking a water tube), for positive reinforcement (e.g., food for food deprived subjects or water for water deprived subjects) according some schedule of reinforcement. Superimposed on this is the classical conditioning component, whereby a CS (e.g., tone or light) is presented and at CS offset a brief electric shock is delivered. This procedure is of interest for a number of reasons. Firstly, it is an early example of the interaction between operant and classical conditioning procedures (Rescorla & Solomon, 1967). Of course, it must be remembered that all operant procedures have classical conditioning contingencies present to some degree. Secondly, the presentation of the tone, after a number of pairings, produced operant response suppression. This was viewed as an indirect measure of a conditioned emotional response, the emotional response being anxiety or fear (Hunt & Brady, 1951; Hebb, 1955; Kamin, 1965). Some researchers believe that there is some similarity between the CER situation and the development of human clinical anxiety (Estes & Skinner, 1941; Wolpe, 1958).

The effects of anxiolytic agents on CER have been inconsistent (Haefely, 1978). Millenson and Leslie (1974)

in their review of CER studies noted minor tranquillers given as an acute dose were effective in attenuating response suppression in nine studies, had no consistent effect in four studies and increased response suppression in one study. Millenson and Leslie (1974) and others (Dantzer, 1977; Huppert & Eversen, 1975) have proposed these inconsistencies result from procedural variations across studies. Caution should be used in interpreting anxiolytic effects on the CER procedure with the best use of the technique being as part of a number of pre-clinical screening tests to obtain an overall behavioural profile of potential anxiolytic agents.

Passive and active avoidance procedures have also been used to examine potential anxiolytic action of psychotropic compounds. Only a few passive avoidance studies have been conducted which have yielded inconsistent results (Davies, et al., 1974; Fuller, 1970; Kumar, 1971a, b). A number of studies have reported depressant effects of anxiolytics at high doses on active avoidance responding (Randall, et al., 1974; Sternbach, et al., 1964) and facilitative effects at low doses (Bignami, 1976; Gray, 1977). Again a number of factors including species, strain and response rate varied across studies making comparisons difficult. Also these factors may modulate anxiolytic action on avoidance responding and help explain the inconsistencies reported in the literature.

Drawing together the results from a number of studies on the behavioural effects of anxiolytics, Haefely (1983) concluded:

> It is generally believed that a valid animal correlate of anxiolytic effect in man ... is the

> attenuation of the disruptive or suppressant effect on spontaneous or operant behavioural responses of (a) response-contingent or non-contingent punishment, (b) fear of punishment, (c) fear of novelty (neophobia) ... and (d) frustration (non-reward). (p. 110).

The approach discussed above is not without its critics. Pinel and Treit (1982) and Treit, et al., (1981) have argued that the use of traditional pre-clinical screening tests are based on arbitrary combinations of stimuli and responses with arbitrary subjects. They proposed an ecologically more valid test was required, citing the work of Seligman (1970) to buttress their proposal. Conditioned defensive burying was such a test according to these researchers.

Pinel and Treit (1978) reported that rats given a single exposure to shock through a wall mounted wire wrapped prod returned to the source of aversive stimulation, the prod, and buried it with bedding material from the floor of the test apparatus. Conditioned defensive burying has now been replicated in a number of studies (Pinel & Treit, 1982) and is regarded as being part of an species specific defense reaction repertoire (Bolles, 1970).

Treit, et al., (1981) examined the conditioned defensive burying response as a rapid screening procedure for anxiolytic agents. They reported that conditioned defensive burying was reduced or eliminated by a number of anxiolytic agents, in a dose-dependent fashion, but was not affected by non-anxiolytic agents. However, we noted a number of methodological weaknesses in their study and refined the procedure. Blampied and Kirk (1983) reported the initial presence of the conditioned defensive burying response but found it was

attenuated completely with diazepam and incompletely with oxprenolol. These changes were independent of interference with initial association of shock and prod, and of changes in general activity. More importantly, we noted conditioned defensive burying extinguished rapidly unlike human anxiety-fear reactions (Levis, 1981). We therefore believe conditioned defensive burying does not represent a particularly useful model for human fear motivated neurotic reactions and that Treit, et al., (1981) have overstated the usefulness of conditioned defensive burying as a screening procedure. It is premature to regard this paradigm as an example of a valid and efficient screening test.

The present experiment examines the anxiolytic action of the benzodiazepine and beta-adrenergic blocker class of psychotropic compoundsusing the context of the passive avoidance methodology introduced in experiment two.

One class of compounds that have recently received attention as having potential as anxiolytic agents are the beta-adrenergic blocking agents (Jefferson, 1974; Noyes, Kathol, Clancy & Crowe, 1981; Pitts & Allen, 1979, 1982; Tyrer, 1976; Whitlock & Price, 1974; Hayes & Schulz, 1983).

Drug compounds can be categorized into two broad types - adrenergic and cholinergic. Adrenergic neurons are those which secrete noradrenaline or other catecholamines at their synapses. Drugs affecting neurotransmitters at such synapses are called adrenergic. Fibres that secrete acetylcholine at their synapses are termed cholinergic and drugs affecting these synapses are cholinergic drugs. The psychophysiology of anxiety-fear is related to the release of certain chemical transmitters within the autonomic nervous system

(ANS); specifically the sympathetic division.

Postganglionic adrenergic innervation of the sympathetic division of the autonomic nervous system is believed to be the site of action of beta-adrenergic blockers. Why are they called beta-blockers? Ahlquist (1948) proposed that there are two types of adrenergic receptors - alpha (α) and beta (β). If the primary response of the innervated organ was excitatory, he termed it an alpha receptor, if it was inhibitory, he termed it a beta receptor. The beta classification has been further subdivided into Beta 1 and Beta 2 receptor types. Beta 1 stimulating or blocking agents act on receptors located in the heart, whereas Beta 2 stimulants or blockers effect receptors located in the blood vessels, kidney and bronchopulmonary areas. The beta blockers are termed blockers because they prevent the binding of noradrenaline to binding sites in the innervated organ.

Beta-adrenergic blockers have a number of properties in addition to beta-adrenergic receptor blockade. Cardioselective beta-blockers, e.g., atenolol and metoprolol, block beta 1 cardiac receptors but will in high doses also block peripheral beta 2 receptors. Non selective beta-blockers, e.g., propranolol, oxprenolol and timolol block both beta 1 and beta 2 receptors. The non selective beta blockers, e.g., propranolol and oxprenolol bind to lipid membranes producing a "local anaesthetic" effect, a membrane stablising activity, but only at high doses. Some beta blockers e.g., oxprenolol, may simultaneously stimulate and block the beta-adrenergic receptor as they have an intrinsic sympathomimetic activity. The clinical effects of these additional properties of beta-adrenergic blockers are not known.

A number of beta-blockers pass through the blood-brain barrier into the cerebrospinal fluid indicating central activity by beta-blockers. A number of beta-adrenergic receptor sites have been identified in the central nervous system. Conway, Greenway and Middlemiss (1978) and Weinstock and Weiss (1980) reported propranolol but not metoprolol or atenolol blocked some central serotonin sites. These findings may have important implications for interpretation of the clinical effects of beta-adrenergic blockers.

Although beta-blockers were introduced for the treatment of cardiovascular disorders recent research endeavour has focused on their potential as anxiolytic agents. According to the Americal Psychiatric Association's DSM - III (Diagnostic and Statistical Manual) anxiety symptoms have both psychological (tension, fear, apprehension, etc.) and somatic (increased heart rate, tremor, sweating, etc.) components. In some anxious patients the autonomic, somatic symptoms are prominent, while in other anxious patients the psychological symptoms of anxiety are the focus of treatment. Hayes and Schulz (1983) have succinctly summarized the complexity of the situation as follows:

> This diversity of anxiety-related symptoms makes it doubtful that anxiety is a single biochemical or physiological abnormality ... It is entirely possible that emotional and physical symptoms are mediated by different neurotransmitters, and that drugs (or a combination of drugs) acting on either one or both systems might be effective in certain subgroups of anxious patients. (p. 104).

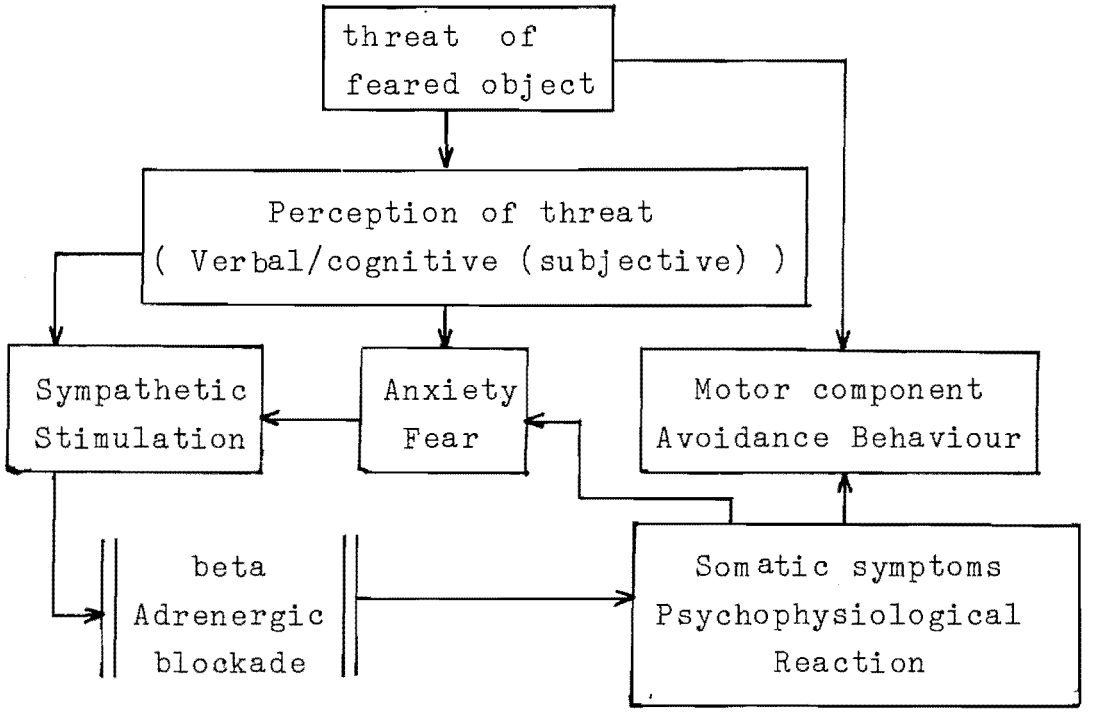
The mechanism of action of beta-blockers in anxiety is not completely clear. They could produce their clinical effect by the blocking of the peripheral beta receptors or

some central effect or an interaction at both levels. Most of the experimental evidence suggests a peripheral beta-adrenergic receptor blocking effect (Hayes & Schulz, 1983).

Lang (1968, 1971) has argued that fear is a complex construct that includes three different systems, the verbal-cognitive (subjective), the motor (behavioural avoidance) and the psycho-physiological. Using Lang's formulation a tentative model of anxiolytic action of beta-adrenergic blockers can be constructed as follows:

Figure 2-4

A tentative model of beta-blocker anxiolytic action.




As stated the somatic symptoms of anxiety-fear include palpitations, tremor, increased heart rate, sweating, etc. which are primarily due to over-activity of the sympathetic division of autonomic nervous system. Preventing the peripheral feedback of the somatic symptoms of anxiety-fear seems to indicate the possibility that beta-adrenergic blockers may have an anxiolytic effect. If indeed this is

the situation, then the use of beta-adrenergic blockers as adjuncts to response prevention treatment or in combination with known anxiolytic agents, e.g., benzodiazepines, should prove a fruitful area of research. Experiment five of this thesis examines the effects of diazepam and propranolol in combination on conditioned licking behaviour.

R.N. Hughes initiated research in our laboratory on the effects of beta-adrenergic blockers on untrained responses, such as, novelty, open field behaviour and emergence test performance. We have continued this research focusing on the effects of beta-adrenergic blockers on trained responses, such as, conditioned defensive burying, passive avoidance, conditioned suppression of drinking and operant appetitive responding. Brought together, the results should enable us to draw up a behavioural profile of beta-adrenergic blockers as potential anxiolytic compounds.

A number of studies have investigated drugs as adjuncts to RP treatment (see introduction). To date the animal research literature has provided little support for the proposition that anxiolytics used with RP will increase the efficacy of RP treatment in hastening avoidance and fear extinction.

The human literature on the use of drugs as adjuncts to exposure treatment of neurotic reactions also contains some disappointing findings, although recent research suggests the use of antidepressants, especially in the treatment of phobic anxiety and panic attacks, shows some promise (Mavissakalian & Barlow, 1981; Zitrin, 1981), with Seehan (1982) advocating their use. In their review of the use of antidepressants in the treatment of agoraphobia, Telch et al.

(1983) noted a number of studies suffered from methodological weaknesses which made conclusions about their results difficult to make. Those studies that were free from such weaknesses questioned the efficacy of such treatments. Similarily, Gray and McPherson (1982) in their review of behavioural treatments of agoraphobia concluded with respect to drug-assisted studies:

> The studies to date combining drugs and behaviour therapy have not been especially encouraging as far as outcome is concerned. (p. 33).

Given the inconsistent findings in both the animal and human literatures on drug assisted exposure treatment for anxiety and fear motivated behaviours the present experiment further examined the use of drugs as adjuncts to RP treatment. Two drug classes were chosen, a benzodinzepine, diazepam and two beta adrenergic blocking agents, propranolol (a non-selective beta-blocker) and atenolol (a cardioselective beta-blocker).

One further factor which has been used in combination with RP treatment is the presence of a non-fearful conspecific with the experimental subject during RP treatment. Baum (1969c) has termed the effect of another nonfearful subject during RP as social facilitation of RP treatment (see introduction chapter).

The presence of a nonfearful conspecific during RP treatment has been described as being analogous to the presence of the therapist during exposure treatment of fear motivated human neurotic behaviours (Baum & Poser, 1971). Indeed, Adams and Hughes (1976) proposed a complete animal analogue of RP treatment consisted of a number of components,

one of which is the presence of another non-anxious subject, a subject regarded as analogous to a therapist.

Epley (1974) reported that fear responses can be attenuated by the presence of a calm conspecific in a variety species, including rodents and humans. A possible explanation for socially mediated fear reduction has been proposed by Moore, Byers and Baron (1981), called the distraction hypothesis. This hypothesis assumes that:

> the more attention a companion elicits the greater the magnitude of the fear reduction response. Factors that are likely to increase the attention devoted to a companion include the companion's novelty and the extent to which the companion is active and engages in social interaction. (p. 486).

In Corriveau et al's., (1978) study only one nonfearful subject was used as the therapist subject across all experimental conditions. Following on from the distraction hypothesis above, it is conceivable that Corriveau et al's., therapist subject was <u>not</u> equally novel, active and engaging in equal amounts of social interaction with the fearful subjects across all experimental conditions. This methodological weakness of their design could have contributed to the absence of a social facilitation effect in the Corriveau et al., (1978) experiment. The present experiment represented an improved research design whereby a different nonfearful subject was used with each fearful experimental subject, thereby equating the conspecifics novelty, activity and social interaction across experimental conditions.

## Method

### Subjects

The subjects are 98 male New Zealand random-bred Wistar rats. These subjects comprised 63 experimentally naive subjects and 35 therapist rats (the non-fearful conspecifics). The experimental subjects age range at the time of testing was 106-213 days with a mean age of 167 days. Their weight range was 229-371 grams, with the mean weight of 259 grams. The therapist rats had an age range of 149-282 days, with a mean age of 204 days. The maintenance schedule was identical to that of experiment one.

### Apparatus

The apparatus was identical to that of experiment two except for the following modifications:

1. The horizontal black and white striped cardboard over the end wall of the right hand 'safe' compartment was removed leaving a stainless steel wall.
2. Overhead compartment illumination was reduced to 50 lux, following pilot work investigating illumination changes on performance.
3. Illumination changes within the apparatus were no longer response dependent. Illumination remained on continuously in the FC compartment and cycled (0.8 sec. off - 0.2 sec. on) in the safe compartment. Delivery of the unconditioned stimulus, shock, and control apparatus was the same as for experiment 2.

Procedure

Prior to the beginning of the experiment, the subjects were randomly allocated to one of nine groups (n=7). An outline of the experimental design is presented in Table 2-12. The experimental procedure was divided into four phases as follows:

1. Habituation. On the first day subjects were placed into the apparatus and allowed to freely move between both compartments for 30 minutes. As with experiment two, the proportional time spent in the FC compartment and approaches into the FC compartment were recorded. A further dependent variable was recorded in this experiment, namely, the latency to the first approach into the FC compartment.

2. Fear Conditioning and Passive Avoidance Acquisition. All groups other than the passive avoidance controls received passive avoidance fear conditioning on day two using the same procedure as experiment two.

3. Response Prevention Treatment. All response prevention subjects received RP treatment for 1 hour in the company of another nonanxious subject, except RP treatment no. therapist controls, RPNT, which received RP treatment alone. In addition to a nonanxious conspecific, RP subjects, except RPT, were assigned to an injection condition, and injected i.p. (intraperitoneal injection) with diazepam, 1.0mg/kg; or propranolol, 10mg/kg; or atenolol, 10mg/kg; or an equivalent volume of 0.9% saline. All injections were in a volume of 1ml/kg. Injections took place 20 minutes before the start of 1 hour RP treatment. Response prevention

TABLE 2-12

Outline of Experimental Design

| Treatment Group | Days (Sessions) | | | | |
|---|---|---|---|---|---|
| | 1(1) | 2(2) | 3 | 4(3) | 5(4) |
| Response prevention control group (RPC) | Hab[a] | PA[b] | HC[d] | Ext[e] | Ext |
| Passive Avoidance control group (PAC) | Hab | AE[c] | HC | Ext | Ext |
| Response prevention with therapist group (RPT) | Hab | PA | RPT | Ext | Ext |
| Response prevention without therapist group (RPNT) | Hab | PA | RPNT | Ext | Ext |
| CS exposure control group - Group (CSC) | Hab | PA | CSC | Ext | Ext |
| Response prevention plus diazepam Group (RPd) | Hab | PA | RPd | Ext | Ext |
| Response prevention plus propranolol Group (RPP) | Hab | PA | RPP | Ext | Ext |
| Response prevention plus atenolol Group (RPA) | Hab | PA | RPA | Ext | Ext |
| Response prevention plus saline control Group (RPS) | Hab | PA | RPS | Ext | Ext |

a Habituation to the apparatus

b Passive avoidance fear conditioning

c Exposure to the apparatus cues without fear conditioning

d Home cage confinement

e Passive avoidance extinction testing

treatment was indentical to that of experiment two.

4. Fear Assessment Measured in Extinction. This phase of the experiment was the same as in experiment two, and experienced by all subjects.

Research Hypotheses

The experimental design generated the following research hypotheses:

1. During habituation on day one all subjects were treated identically therefore should show no difference in (i) the time spent in the FC compartment, (ii) the number of approaches into the FC compartment and (iii) the latency to first enter the FC compartment.

2(a) During session two all groups, except PAC, received passive avoidance fear conditioning. If passive avoidance fear conditioning motivated subjects to avoid entry into the FC compartment, then in comparison to PAC subjects, they should (i) spend less cumulative time in the FC compartment, and (ii) make fewer approaches into the FC compartment.

2(b) If passive avoidance fear conditioning had the same effects on all subjects, then these subjects should show no difference in (i) the cumulative time spent in the FC compartment, and (ii) the number of approaches into the FC compartment.

3(a) If RP treatment reduced fear associated with the FC compartment then Group RPNT subjects in comparison to Group RPC subjects should (i) spend more cumulative time in the FC compartment, (ii) make more approaches into the FC compartment and (iii) have a shorter latency to first enter

the FC compartment.

3(b) If RP treatment has completely eliminated fear of the FC compartment then RP treated subjects in comparison to PAC subjects should show no difference in (i) the cumulative time spent in the FC compartment, (ii) the number of approaches into the FC compartment and (iii) the latency to first enter the FC compartment.

3(c) If passive avoidance fear conditioning continues to motivate subjects to avoid entry into the FC compartment across sessions, then PAC subjects in comparison to RPC subjects should (i) spend more cumulative time in the FC compartment, (ii) make more approaches into the FC compartment and (iii) have a shorter latency to first enter the FC compartment.

3(d) If the presence of a non-fearful conspecific during RP treatment facilitated fear reduction then RPT subjects in comparison to RPNT subjects should (i) spend more cumulative time in the FC compartment, (ii) make more approaches into the FC compartment and (iii) have a shorter latency to first enter the FC compartment.

3(e) If forced non-reinforced CS exposure (response prevention) is more effective in reducing conditioned fear than free non reinforced CS exposure, flooding, then RPNT subjects in comparison to CSC subjects should (i) spend more cumulative time in the FC compartment, (ii) make more approaches into the FC compartment and (iii) have a shorter latency to first enter the FC compartment.

3(f) If drug assisted RP treatment is more effective in reducing conditioned fear than RP treatment per se, then RPd, RPP and RPA subjects in comparison to RPS subjects

should (i) spend more cumulative time in the FC compartment, (ii) make more approaches into the FC compartment and (iii) have a shorter latency to first enter the FC compartment.

4. If over the passive avoidance extinction session conditioned fear occurs then RP treated subjects in comparison to PAC subjects should show no difference in (i) the cumulative time spent in the FC compartment, (ii) the number of approaches into the FC compartment and (iii) the latency to first enter the FC compartment.

## Results and Discussion

As for experiment two, the raw scores yielded by the dependent variables were treated in the following way.

1. The total voluntary time spent in the FC compartment was converted to proportion of session time in the FC compartment.

2. Number of approaches into the FC compartment was converted to a rate measure, approaches per minute.

3. The latency to first enter the FC compartment was taken as the time in seconds from the beginning of each session to move into the FC compartment for the first time. A subject failing to enter the FC compartment was given a latency of 1800 seconds. Because some subjects had short latency scores and others long latency scores the latency to first enter the FC compartment scores exhibited heterogeneity of variance which was alleviated by a log transformation. The transformed scores were used in the following data analyses.

### Time Spent in the FC Compartment

Group means and standard deviations across sessions are presented in Table 2-13, with the means being graphically

TABLE 2-13

Means and standard deviations of proportional time spent in the FC compartment.

| Treatment Group | | Sessions | | | | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | Overall |
| RPT | M | 0.401 | 0.167 | 0.390 | 0.363 | 0.330 |
| | SD | 0.086 | 0.050 | 0.291 | 0.299 | 0.225 |
| RPNT | M | 0.379 | 0.130 | 0.100 | 0.180 | 0.197 |
| | SD | 0.034 | 0.044 | 0.071 | 0.169 | 0.143 |
| PAC | M | 0.443 | 0.519 | 0.507 | 0.489 | 0.489 |
| | SD | 0.080 | 0.092 | 0.052 | 0.105 | 0.085 |
| RPC | M | 0.434 | 0.149 | 0.004 | 0.004 | 0.148 |
| | SD | 0.061 | 0.030 | 0.005 | 0.008 | 0.182 |
| CSC | M | 0.463 | 0.169 | 0.080 | 0.144 | 0.214 |
| | SD | 0.047 | 0.071 | 0.095 | 0.167 | 0.180 |
| RPS | M | 0.430 | 0.187 | 0.397 | 0.424 | 0.360 |
| | SD | 0.069 | 0.054 | 0.270 | 0.186 | 0.190 |
| RPd | M | 0.477 | 0.144 | 0.396 | 0.464 | 0.370 |
| | SD | 0.083 | 0.055 | 0.306 | 0.385 | 0.273 |
| RPP | M | 0.383 | 0.130 | 0.766 | 0.327 | 0.401 |
| | SD | 0.091 | 0.024 | 0.292 | 0.304 | 0.311 |

Table 2.13 Cont.

| Treatment Group | | Sessions | | | | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | Overall |
| RPA | M | 0.440 | 0.171 | 0.377 | 0.250 | 0.310 |
| | SD | 0.060 | 0.065 | 0.257 | 0.165 | 0.184 |
| Overall | M | 0.428 | 0.196 | 0.335 | 0.294 | |
| | SD | 0.073 | 0.128 | 0.304 | 0.261 | |

represented in figure 2-5. A split-plot ANOVA revealed that the time spent in the FC compartment was affected by group membership, ($F(8,54) = 7.53$, $p<.001$), by session number, ($F(3,162) = 28.26$, $p<.001$), and by their interaction, ($F(24,162) = 5.91$, $p<.001$).

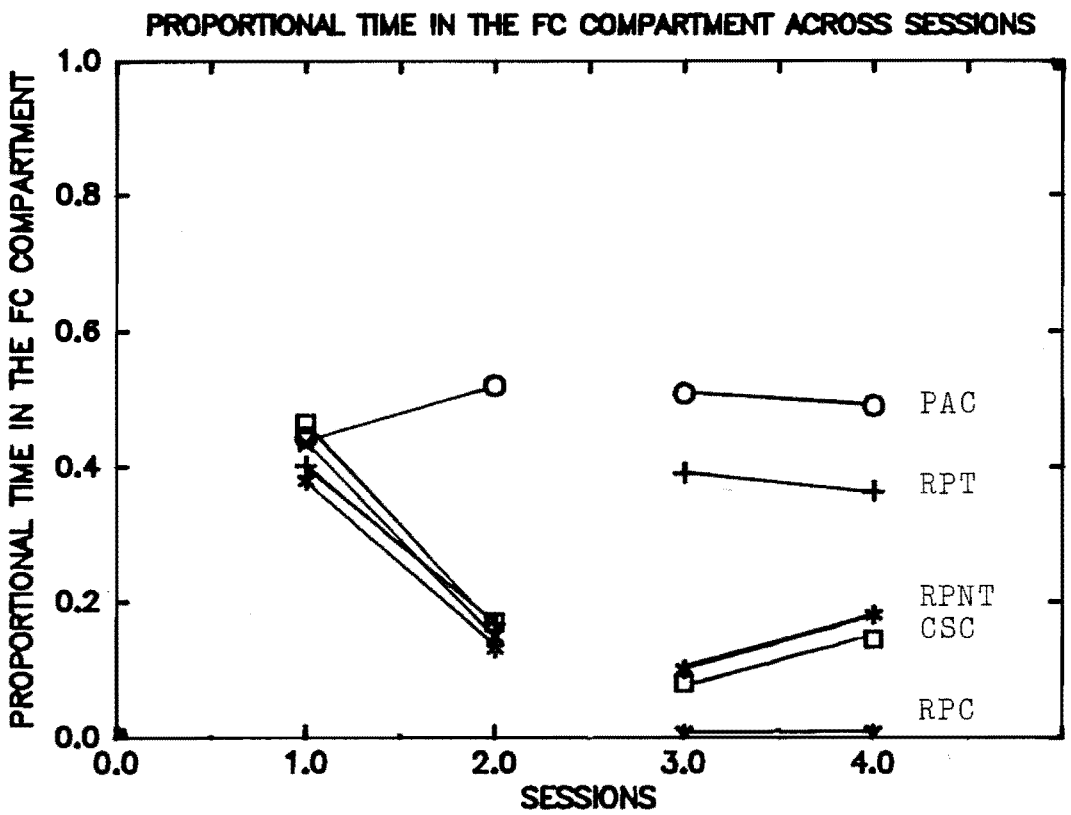
As in experiment two, groups spent an equivalent amount of time in the FC compartment during session one, ($F(8,54) = 1.63$, $p>.05$). Overall, subjects spent 42.8% of the first session in the FC compartment indicating a slight bias to the safe compartment.

The time spent in the FC compartment varied between groups, ($F(8,54) = 31.95$, $p<.001$), during session two. Tukey multiple comparison tests, summarized in table 2-14, confirmed that all subjects receiving fear conditioning significantly reduced time spent in the FC compartment. As expected, there was no difference in the time measure by PAC subjects between sessions one and two. During session two, PAC subjects spent 51.9% of the session time in the FC compartment in comparison to only 15.6% by all other subjects. Tukey multiple comparison tests, summarized in table 2-15, confirmed that the fear conditioning procedure produced an equivalent amount of avoidance of the FC compartment during session two.

Extinction of passive avoidance responding was examined during sessions three and four. Passive avoidance control subjects divided their time equally between compartments over the two extinction sessions, spending 50.7% and 48.9% of the sessions in the FC compartment. On the other hand, response prevention control subjects spent very little time, 0.4%, in the FC compartment during each extinction session.

FIGURE 2-5 Proportional time spent in the FC compartment for each group across sessions.

2-5 (a)




2-5 (b)

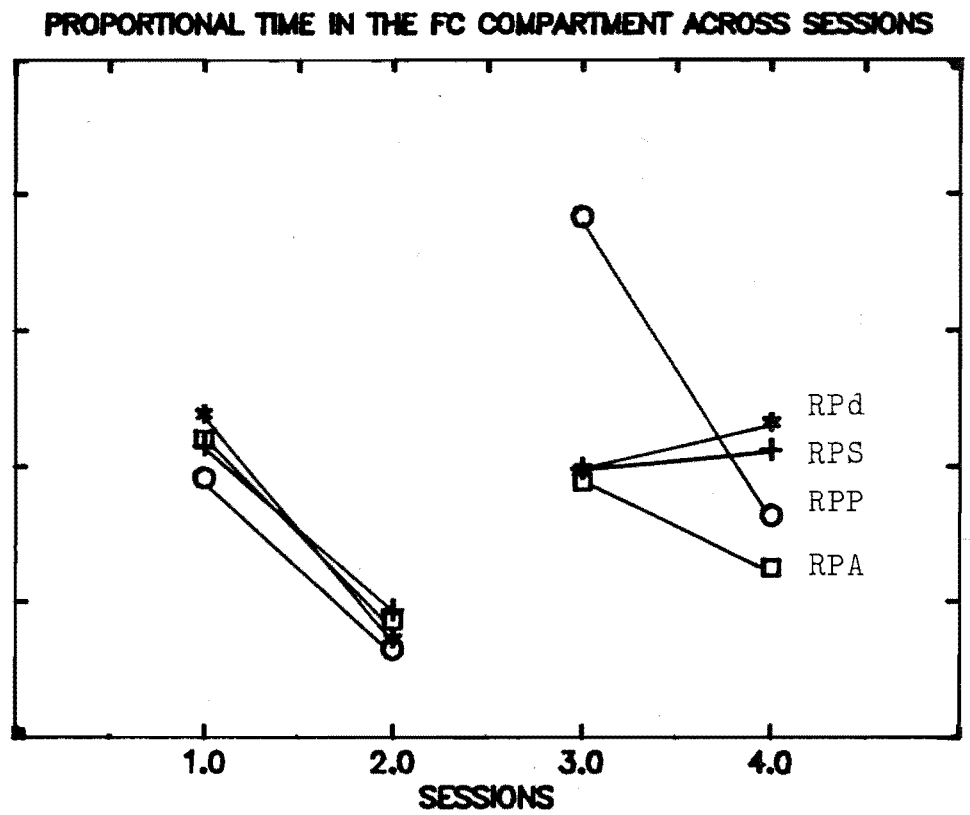


TABLE 2-14

Summary of the Tukey comparison tests for the Sessions Factor at each level of the Groups Factor : Proportional time data.

| Treatment Group | Session Comparisons | | | | | |
|---|---|---|---|---|---|---|
| | 1-2 | 1-3 | 1-4 | 2-3 | 2-4 | 3-4 |
| RPT | 4.333 | NS | NS | 4.130 | NS | NS |
| RPNT | 4.611 | 5.167 | 3.685 | NS | NS | NS |
| PAC | NS[1] | NS | NS | NS | NS | NS |
| RPC | 5.278 | 7.963 | 7.963 | NS | NS | NS |
| CSC | 5.444 | 7.092 | 5.907 | NS | NS | NS |
| RPS | 4.500 | NS | NS | 3.889 | 4.389 | NS |
| RPd | 6.167 | NS | NS | 4.667 | 5.926 | NS |
| RPP | 4.685 | 7.093 | NS | 11.778 | NS | 8.130 |
| RPA | 4.981 | NS | NS | 3.815 | NS | NS |

df = 4, 162

q.05 = 3.66

q.01 = 4.48

[1]NS = non-significant comparison

TABLE 2-15

Summary of the Tukey comparison tests for the Groups Factor as each level of the Sessions Factor : Proportional time data.

| Group Comparisons | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| RPC-PAC | NS[1] | 6.076 | 8.259 | 7.964 |
| RPT-RPNT | NS | NS | 4.762 | 3.005 |
| RPNT-CSC | NS | NS | NS | NS |
| RPT-CSC | NS | NS | 5.090 | 3.596 |
| RPC-RPNT | NS | NS | NS | 2.890 |
| RPC-RPT | NS | NS | 6.338 | 5.895 |
| RPC-CSC | NS | NS | NS | NS |
| RPC-RPS | NS | NS | 6.453 | 6.897 |
| RPC-RPd | NS | NS | 6.437 | 7.553 |
| RPC-RPP | NS | NS | 12.512 | 5.304 |
| RPC-RPA | NS | NS | 6.125 | 4.039 |
| PAC-RPNT | NS | 6.387 | 6.683 | 5.074 |
| PAC-RPT | NS | 5.780 | NS | NS |
| PAC-CSC | NS | 5.747 | 7.011 | 5.665 |
| PAC-RPS | NS | 5.452 | NS | NS |
| PAC-RPd | NS | 6.158 | NS | NS |
| PAC-RPP | NS | 6.387 | 4.253 | NS |
| PAC-RPA | NS | 5.714 | NS | 3.924 |

Table 2-15 Cont.

| Group Comparisons | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| RPS-RPd | NS | NS | NS | NS |
| RPS-RPP | NS | NS | 6.059 | NS |
| RPS-RPA | NS | NS | NS | NS |
| RPd-RPP | NS | NS | 6.076 | NS |
| RPd-RPA | NS | NS | NS | 3.514 |
| RPP-RPA | NS | NS | 6.387 | NS |

$q'_{.05} = 2.811$

$q'_{.01} = 3.730$

[1]NS = non-significant comparison

The difference in performance between PAC and RPC subjects can be attributed to the effect of fear conditioning received by the RPC subjects.

Performance of the response prevention treated subjects over the extinction sessions indicated that RP significantly reduced avoidance and fear associated with the FC compartment (refer tables 2-14 and 2-15). Response prevention treated subjects increased their time spent in the FC compartment from session two (15.5%) to session three (40.4%) and session four (33.5%). On the other hand, RPC control subjects' time spent in the FC compartment decreased from 14.9% during session two to 0.4% during sessions three and four.

To examine the hypothesis that social facilitation would increase the efficacy of RP treatment Tukey comparison tests were conducted between RPNT and RPT subjects across sessions three and four. Response prevention in the presence of a non-fearful conspecific significantly produced greater fear reduction and less avoidance of the FC compartment in comparison to RP alone (refer to table 2-15). Across extinction sessions RPT subjects spent 36.5% of the session time in the FC compartment in comparison to 14.0% of the session time for RPNT subjects. Clearly, response prevention treatment can be enhanced by socially mediated fear reduction.

To examine the hypothesis that drugs would increase the efficacy of RP treatment Tukey comparison tests were conducted between RPS, RPd, RPP and RPA subjects across session three and four. The hypothesis was not supported except for subjects injected with propranolol prior to RP treatment (refer table 2-15). Across sessions three and four, saline-RP treated subjects spent 41.0% of the session time in the

FC compartment, while diazepam-RP treated subjects spent slightly more time in the FC compartment, 43.0%. Only propranolol-RP treated subjects spent significantly more time in the FC compartment, 54.6%, incomparison to saline-RP treated controls.

If RP treatment completely eliminated fear associated with the FC compartment, there should be no difference in the time measure between PAC and RP treatment subjects. Except for RPNT subjects which showed some evidence of residual fear following RP treatment, RP treatment with another non-fearful conspecific completely eliminated conditioned fear and avoidance of the FC compartment (refer tables 2-14 and 2-15).

Approaches into the FC Compartment

Group means and standard deviations across sessions are presented in table 2-16, with the means being graphically represented in figure 2-6.

A split-plot ANOVA revealed that approaches into the FC compartment were significantly affected by group membership, ($F(8,54) = 4.96$, $p<.001$), session number, ($F(3,162) = 71.95$, $p<.001$) and their interaction, ($F(24,162) = 2.64$, $p<.001$).

In contrast to the findings of experiment two, approaches into the FC compartment varied between groups during session one, ($F(5,84) = 3.10$, $p<.01$). Tukey multiple group comparisons, summarized in table 2-17 confirmed that the variation was primarily the result of a high number of approaches per minute by RPA (3.82) and CSC (3.4) groups in comparison to RPT (2.5) and RPC (2.2) groups.

Approaches into the FC compartment significantly varied

TABLE 2-16

Means and standard deviations of approaches per minute into the FC compartment.

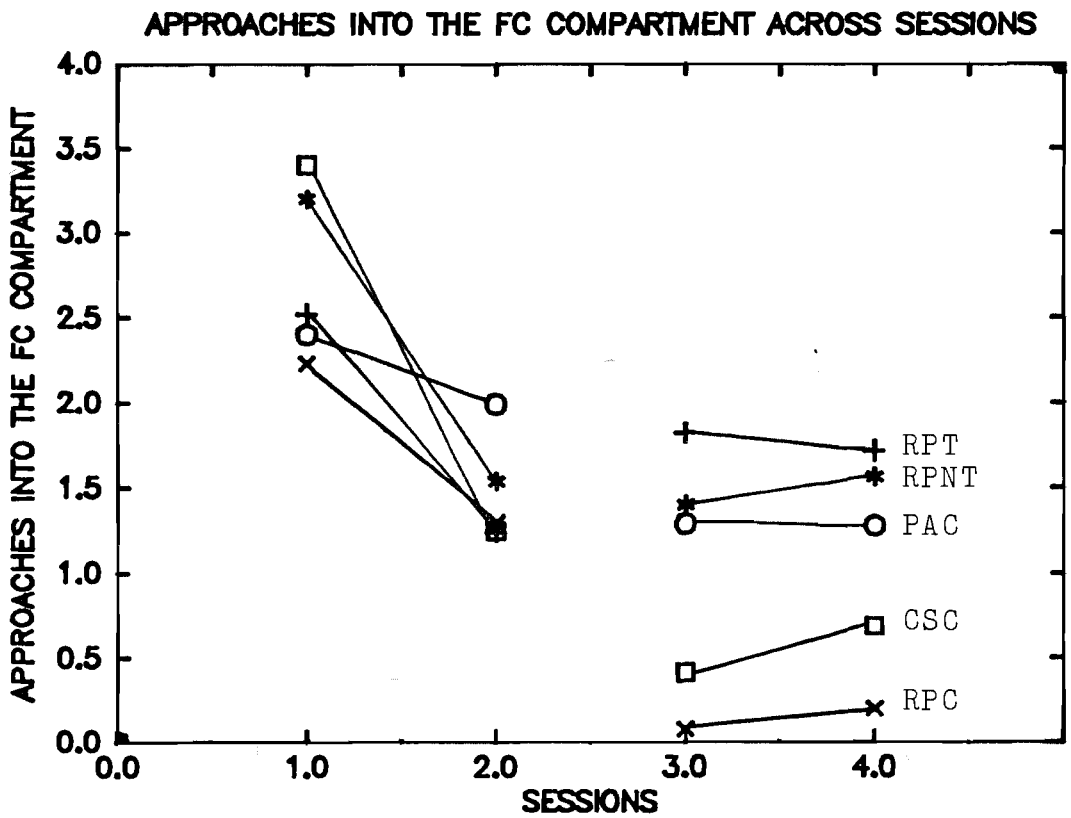
| Treatment Group | | Sessions | | | | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | Overall |
| RPT | M | 2.521 | 1.241 | 1.820 | 1.713 | 1.824 |
| | SD | 0.520 | 0.720 | 1.188 | 1.215 | 1.017 |
| RPNT | M | 3.197 | 1.537 | 1.397 | 1.560 | 1.923 |
| | SD | 0.875 | 0.377 | 0.655 | 0.888 | 1.019 |
| PAC | M | 2.400 | 1.989 | 1.287 | 1.274 | 1.738 |
| | SD | 0.802 | 0.685 | 0.484 | 0.445 | 0.763 |
| RPC | M | 2.230 | 1.297 | 0.076 | 0.197 | 0.950 |
| | SD | 0.759 | 0.283 | 0.099 | 0.349 | 0.988 |
| CSC | M | 3.400 | 1.246 | 0.413 | 0.686 | 1.436 |
| | SD | 1.004 | 0.545 | 0.481 | 0.879 | 1.393 |
| RPS | M | 2.786 | 1.494 | 1.651 | 1.757 | 1.922 |
| | SD | 0.905 | 0.716 | 0.581 | 0.646 | 0.855 |
| RPd | M | 2.537 | 0.999 | 1.563 | 1.040 | 1.535 |
| | SD | 0.556 | 0.458 | 1.179 | 0.384 | 0.925 |
| RPP | M | 2.817 | 1.593 | 2.123 | 1.486 | 2.005 |
| | SD | 0.374 | 0.525 | 1.115 | 1.144 | 0.974 |

Table 2.16 Cont.

| Treatment Group | | Sessions | | | | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | Overall |
| RPA | M | 3.819 | 1.780 | 2.550 | 1.830 | 2.495 |
| | SD | 0.971 | 0.435 | 1.598 | 0.951 | 1.312 |
| Overall | M | 2.856 | 1.464 | 1.431 | 1.283 | |
| | SD | 0.879 | 0.587 | 1.143 | 0.931 | |

FIGURE 2-6 Approaches per minute into the FC compartment for each group across sessions.

2-6 (a)




2-6 (b)

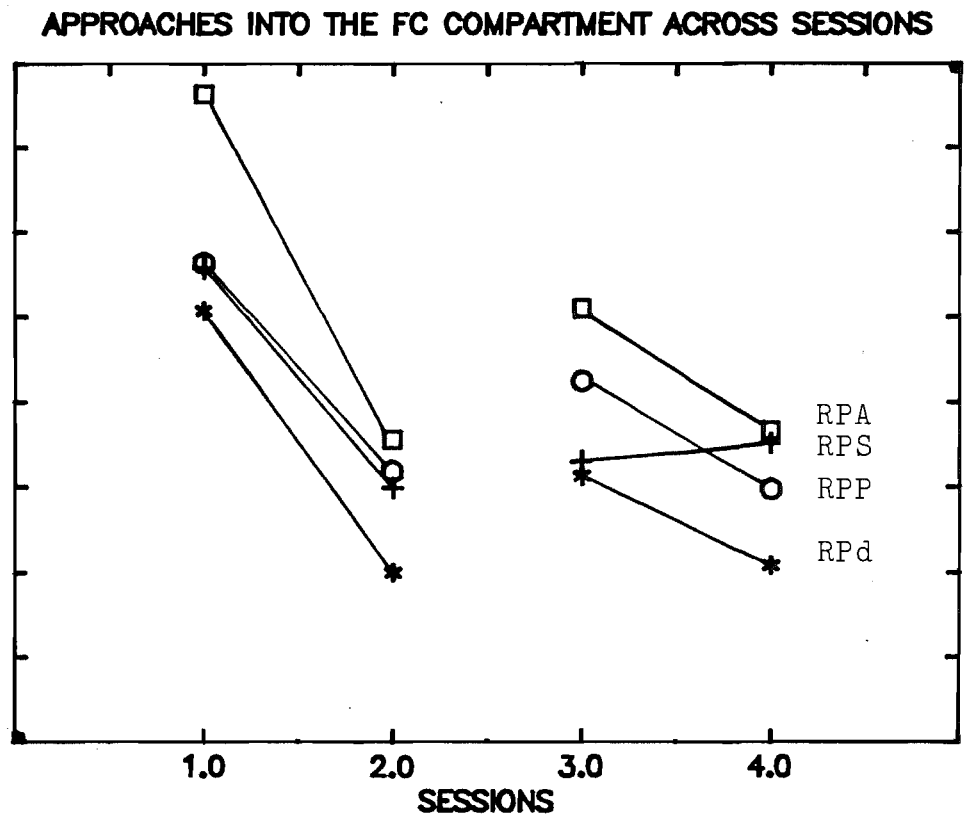


TABLE 2-17

Summary of the Tukey comparison tests for the Groups Factor at each level of the Sessions Factor : Approaches into the FC compartment data.

| Group Comparisons | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| RPC-PAC | NS[a] | NS | 4.091 | 3.638 |
| RPT-RPNT | NS | NS | NS | NS |
| RPT-CSC | 2.969 | NS | 4.753 | 3.470 |
| RPNT-CSC | NS | NS | 3.324 | 2.953 |
| RPC-RPT | NS | NS | 5.892 | 5.122 |
| RPC-RPNT | 3.267 | NS | 4.463 | 4.605 |
| RPC-CSC | 3.953 | NS | NS | NS |
| RPC-RPS | NS | NS | 5.321 | 5.270 |
| RPC-RPd | NS | NS | 5.024 | NS |
| RPC-RPP | NS | NS | 6.915 | 4.355 |
| RPC-RPA | 5.368 | NS | 8.358 | 5.517 |
| PAC-RPT | NS | NS | NS | NS |
| PAC-RPNT | NS | NS | NS | NS |
| PAC-CSC | 3.378 | NS | 2.953 | NS |
| PAC-RPS | NS | NS | NS | NS |
| PAC-RPd | NS | 3.344 | NS | NS |
| PAC-RPP | NS | NS | 2.824 | NS |
| PAC-RPA | 4.794 | NS | 4.267 | NS |

Table 2-17 Cont.

| Group Comparisons | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| RPS-RPd | NS | NS | NS | NS |
| RPS-RPP | NS | NS | NS | NS |
| RPS-RPA | 3.490 | NS | 3.037 | NS |
| RPd-RPP | NS | NS | NS | NS |
| RPd-RPA | 4.331 | NS | 3.334 | NS |
| RPP-RPA | 3.385 | NS | NS | NS |

$q'.05 = 2.811$

$q'.01 = 3.731$

a = non-significant comparison

between groups during session two, ($F(8,54) = 2.16$, $p<.05$). Tukey multiple comparison tests, summarized in table 2-18, confirmed that fear conditioning significantly reduced approaches into the FC compartment during session two. Passive avoidance control subjects also decreased approaches into the FC compartment during session two, but this decrease was non-significant (refer table 2-18). While groups receiving fear conditioning significantly decreased approaches into the FC compartment during session two the number of approaches were not significantly lower, except for RPd subjects, than the approaches of PAC subjects. As in experiment two, the approaches dependent variable exhibited more variability in comparison to the time measure.

Across extinction sessions three and four PAC subjects' approaches per minute remained constant, (1.28 and 1.27, respectively). In contrast, RPC subjects reduced approaches from session two (1.297) to session three (0.076) and increased approaches from session three to session four (0.197). All these changes were statistically significant (refer table 2-18). Also approaches into the FC compartment during the extinction sessions were significantly less for RPC subjects in comparison to PAC subjects (refer table 2-17). This indicated once again that fear of the FC compartment was persistent over time, with little extinction of conditioned fear taking place with the RPC subjects.

The number of approaches into the FC compartment over the extinction sessions by response prevention treated subjects was significantly more than that of the RPC subjects (refer table 2-17). Response prevention treatment reduced conditioned fear and avoidance of the FC compartment when assessed using

TABLE 2-18

Summary of the Tukey comparison tests for the Sessions Factor at each level of the Groups Factor : Approaches into the FC compartment data.

| Treatment Group | Sessions comparison | | | | | |
|---|---|---|---|---|---|---|
| | 1-2 | 1-3 | 1-4 | 2-3 | 2-4 | 3-4 |
| RPT | 4.923 | NS[1] | NS | NS | NS | NS |
| RPNT | 6.385 | 6.923 | 6.296 | NS | NS | NS |
| PAC | NS | 4.281 | 4.331 | NS | NS | NS |
| RPC | NS | 8.285 | 7.819 | 4.696 | 4.231 | NS |
| CSC | 8.285 | 11.488 | 10.438 | NS | NS | NS |
| RPS | 4.969 | 4.365 | 3.958 | NS | NS | NS |
| RPd | 5.915 | 3.746 | 5.758 | NS | NS | NS |
| RPP | 4.708 | NS | 5.119 | NS | NS | NS |
| RPA | 7.842 | 4.881 | 7.650 | NS | NS | NS |

df = 4, 162

q.05 = 3.66

q.01 = 4.48

[1]NS = non-significant comparison

the approaches fear assessment measure.

The social facilitation effect was found with the time measure, but was not found with the approaches measure (refer table 2-17). There was no significant difference between RPT and RPNT subjects over extinction sessions in approaches into the FC compartment, with the mean number of approaches per minute for RPT subjects being 1.77 and for the RPNT subjects, 1.48. This finding illustrates again the greater variability of the approaches measure, but also must question the robustness of the social facilitation effect.

The approaches measure failed to discern an increase in efficacy of RP treatment by diazepam, propranolol or atenolol Once again RP was not facilitated by the concurrent use of anxiolytic agents. Whereas propranolol had an efficacious effect when assessed by the time fear assessment measure, it had no effect when assessed by the approaches fear assessment measure. This raises the possibility that these two fear assessment measures are sensitive to different aspects of the same phenomonon, conditioned fear. It is possible that drug assisted RP effects are sensitively measured by the time measure, but not by the insensitive approaches measure.

Latency to First Enter the FC Compartment

This dependent variable has been shown to be a sensitive fear assessment measure when the conditioned avoidance response was step-down passive avoidance (Corriveau & Smith, 1978). The present experiment examined this measure with a passive avoidance shuttle response.

Group means and standard deviations across sessions are presented in table 2-19, with the means being graphically

TABLE 2-19

Means and standard deviations of the log latency to first enter the FC compartment.

| Treatment Group | | Sessions | | | | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | Overall |
| RPT | M | 1.080 | 0.650 | 0.443 | 0.954 | 0.782 |
| | SD | 0.291 | 0.280 | 0.350 | 0.375 | 0.400 |
| RPNT | M | 1.080 | 0.520 | 1.067 | 1.101 | 0.942 |
| | SD | 0.389 | 0.185 | 0.380 | 0.436 | 0.421 |
| PAC | M | 0.937 | 0.551 | 0.497 | 0.426 | 0.603 |
| | SD | 0.243 | 0.196 | 0.254 | 0.242 | 0.299 |
| RPC | M | 0.994 | 0.633 | 2.279 | 2.447 | 1.588 |
| | SD | 0.128 | 0.231 | 1.260 | 0.735 | 1.064 |
| CSC | M | 0.893 | 0.576 | 2.026 | 2.309 | 1.451 |
| | SD | 0.302 | 0.468 | 1.209 | 1.143 | 1.114 |
| RPS | M | 1.036 | 0.863 | 0.899 | 1.101 | 0.974 |
| | SD | 0.266 | 0.235 | 0.374 | 0.635 | 0.398 |
| RPd | M | 0.796 | 0.334 | 0.928 | 1.120 | 0.794 |
| | SD | 0.273 | 0.245 | 0.603 | 0.625 | 0.533 |
| RPP | M | 0.927 | 0.674 | 1.067 | 1.919 | 1.147 |
| | SD | 0.277 | 0.515 | 0.962 | 1.232 | 0.919 |

Table 2-19 Cont.

| Treatment Group | | Sessions 1 | 2 | 3 | 4 | Overall |
|---|---|---|---|---|---|---|
| RPA | M | 0.914 | 0.523 | 0.511 | 0.821 | 0.692 |
| | SD | 0.277 | 0.321 | 0.425 | 0.397 | 0.385 |
| Overall | M | 0.962 | 0.591 | 1.080 | 1.355 | |
| | SD | 0.275 | 0.326 | 0.934 | 0.947 | |

represented in figure 2-7.

A split-plot ANOVA revealed that the latency measure was significantly affected by group membership, ($F(8,54) = 6.22$, $p<.001$), session number, ($F(3,162) = 25.6$, $p<.001$), and by their interaction, ($F(24,162) = 4.65$, $p<.001$).

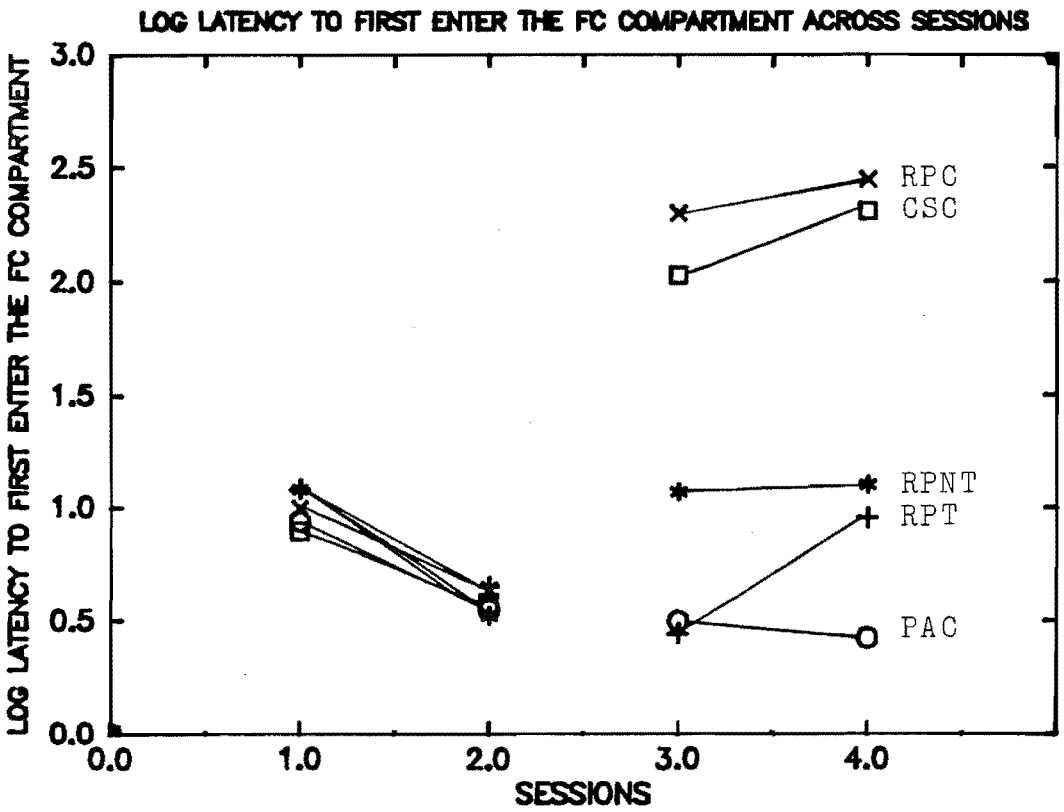
The latency measure was constant between groups during session one, ($F(8,54)< 1$, $p>.05$), and session two, ($F(8,54 = 1.42$, $p>.05$). Because the latency measure is recorded before fear conditioning during session two, this measure can only be used to assess the effects of fear conditioning in sub sequent sessions.

The latency measure varied across groups during session three, ($F(8,54) = 5.45$, $p<.001$) and session four, ($F(8,54) = 6.54$, $p<.001$). Passive avoidance control subjects had a mean log latency to first enter the FC compartment during session three of 0.497 (3.14 seconds) and during session four 0.426 (2.67 seconds). In comparison, RPC subjects had a mean log latency during session three of 2.279 (190 seconds) and during session four of 2.447 (280 seconds). While the latency measure decreased across extinction sessions for PAC subjects it increased for RPC subjects, indicating RPC subjects were spending more time in the safe compartment before entering the FC compartment in comparison to subjects which did not receive fear conditioning. Clearly, the latency to first enter the FC compartment is sensitive to the effects of fear conditioning.

Performance of the RP treated subjects over the extinction sessions indicated that RP significantly reduced avoidance and conditioned fear associated with the FC compartment (refer tables 2-20 and 2-21). The mean latency to first enter

FIGURE 2-7 Log latency to first enter the FC compartment for each group across sessions

2-7 (a)




2-7 (b)

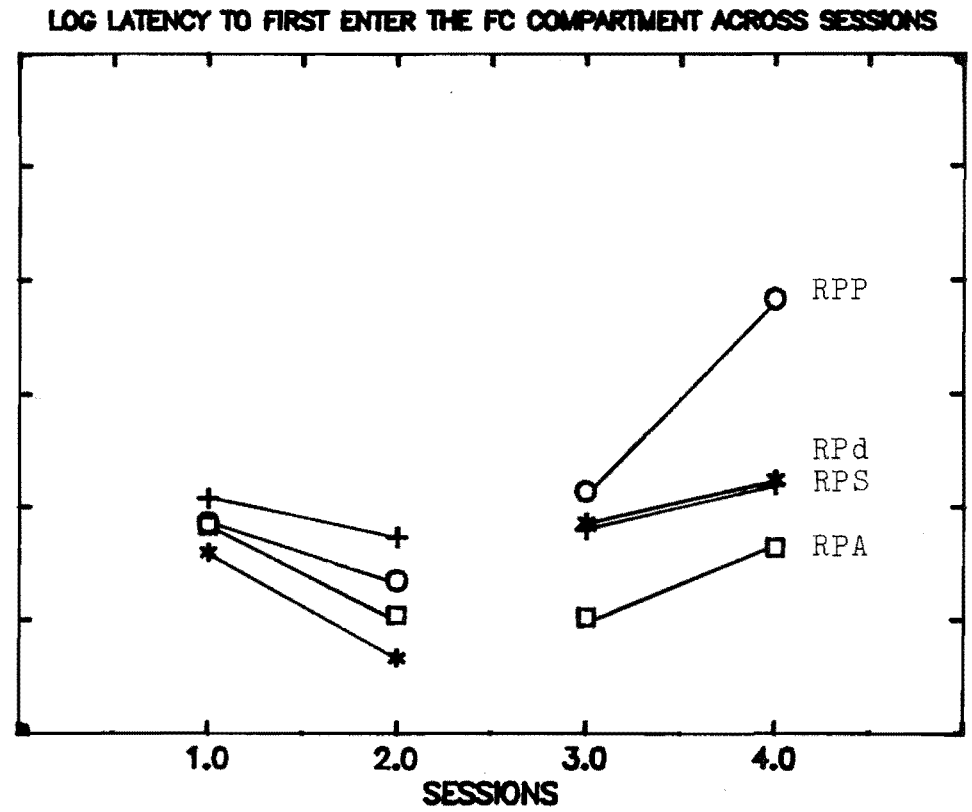


TABLE 2-20

Summary of the Tukey comparison tests for the Groups Factor as each level of the Sessions Factor : Latency to first enter the FC compartment data.

| Group Comparisons | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| RPC-PAC | NS[1] | NS | 8.414 | 9.542 |
| RPT-RPNT | NS | NS | 2.946 | NS |
| RPT-CSC | NS | NS | 7.474 | 6.397 |
| RPNT-CSC | NS | NS | 4.528 | 5.703 |
| RPC-RPT | NS | NS | 8.668 | 7.049 |
| RPC-RPNT | NS | NS | 5.722 | 6.355 |
| RPC-CSC | NS | NS | NS | NS |
| RPC-RPS | NS | NS | 6.516 | 6.355 |
| RPC-RPd | NS | NS | 6.379 | 6.265 |
| RPC-RPP | NS | NS | 5.722 | NS |
| RPC-RPA | NS | NS | 8.347 | 7.677 |
| PAC-RPT | NS | NS | NS | NS |
| PAC-RPNT | NS | NS | NS | 3.187 |
| PAC-CSC | NS | NS | 7.219 | 8.890 |
| PAC-RPS | NS | NS | NS | 3.187 |
| PAC-RPd | NS | NS | NS | 3.277 |
| PAC-RPP | NS | NS | NS | 7.049 |
| PAC-RPA | NS | NS | NS | NS |

Table 2-20 Cont.

| Group Comparisons | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| RPS-RPd | NS | NS | NS | NS |
| RPS-RPP | NS | NS | NS | 3.862 |
| RPS-RPA | NS | NS | NS | NS |
| RPd-RPP | NS | NS | NS | 3.772 |
| RPd-RPA | NS | NS | NS | NS |
| RPP-RPA | NS | NS | NS | 5.184 |

$q'.05 = 2.810$

$q'.01 = 3.729$

[1]NS = non-significant comparison

TABLE 2-21

Summary of the Tukey comparison tests for the Sessions Factor at each level of the Groups Factor : Latency to first enter the FC compartment data.

| | Session comparisons | | | | | |
|---|---|---|---|---|---|---|
| Treatment Group | 1-2 | 1-3 | 1-4 | 2-3 | 2-4 | 3-4 |
| RPT | NS[1] | NS | NS | NS | NS | NS |
| RPNT | NS | NS | NS | NS | NS | NS |
| PAC | NS | NS | NS | NS | NS | NS |
| RPC | NS | 6.842 | 7.737 | 8.965 | 9.659 | NS |
| CSC | NS | 6.033 | 7.540 | 7.721 | 9.228 | NS |
| RPS | NS | NS | NS | NS | NS | NS |
| RPd | NS | NS | NS | NS | 4.185 | NS |
| RPP | NS | NS | 5.282 | NS | 6.629 | 4.537 |
| RPA | NS | NS | NS | NS | NS | NS |

df = 4, 162

q.05 = 3.66

q.01 = 4.48

[1]NS = non-significant comparison

the FC compartment was significantly less for RP subjects (9.88 seconds) in comparison to RPC subjects (235 seconds) over the extinction sessions (refer to table 2-20). While the latency measure significantly increased across sessions for RPC subjects it remained relatively constant for RP treated subjects (refer table 2-21).

As with the time measure, the latency measure indicated that CSC subjects performed more like RPC subjects than RP treated subjects. The mean latency to first enter the FC compartment over the extinction sessions for CSC subjects was 147 seconds which compares with 235 seconds for RPC subjects and 9.88 seconds for RP treated subjects. The CSC subjects increased their latency to first enter the FC compartment over sessions, as did RPC subjects, (refer table 2-21) and did not significantly differ from RPC subjects across sessions, (refer table 2-20). It is clear that giving subjects the opportunity to explore and enter the FC compartment without preventing escape, a flooding procedure, was not sufficient to produce significant conditioned fear reduction in comparison to the response prevention procedure.

Partial support for the social facilitation effect was obtained with the latency measure. The latency to first enter the FC compartment was significantly less for RPT subjects (2.77 seconds) during session three than for RPNT subjects (11.66 seconds), refer to table 2.20. But for session four, this difference had disappeared, mainly due to RPT subjects increasing the latency to 9.0 seconds, with RPNT subjects' latency remaining about the same as for session three at 12.62 seconds. Why RPT subjects should increase their latencies to first enter the FC compartment

from session three to session four is not known. It is possible that inter subject variability accounted for some of this result. Examination of individual subject scores indicated all subjects, except one, had short latencies to first enter the FC compartment. One subject had a latency of 41 seconds which lifted the mean latency for RPT subjects to that of RPNT subjects during session four.

The latency measure failed to demonstrate an increase in efficacy of RP treatment by diazepam, propranolol or atenolol, as indicated by non-significant Turkey comparison tests between RPS subjects and RPd, RPP and RPA subjects reported in table 2-20. While RPS, RPA and RPd subjects had very similar latencies, the latency for RPP subjects significantly increased from session three to session four (refer to table 2-21). Again, inter subject variability may account, in part, for this result. Four out of the seven RPP subjects had very high latencies to first enter the FC compartment during session four, namely, 1610, 1659, 1105 and 1487 seconds. The remaining subjects had relatively short latencies, namely, 30, 5 and 30 seconds.

If RP completely eliminated fear associated with the FC compartment, the latency to first enter the FC compartment should not significantly differ between PAC and RP treated subjects. This relationship was found for session three but not for session four latencies (refer table 2-20). That is, there was no evidence of residual fear during session three, according to the latency measure, but, some evidence of it during session four. This result can be explained by the finding that all RP treated groups had increases in the latency measure from session three to session four, most

dramatically seen with RPP subjects (refer figure 2-7), while PAC subjects decreased their mean latency from session three to session four. Even though this result is a little puzzling, it should be noted that RP treated subjects were still entering the FC compartment for the first time with significantly shorter latencies than RPC subjects. That is, overall RP treatment facilitated initial entry into the FC compartment in comparison to control subjects.

As with the other two fear assessment methods, the latency measure for the apparatus exposure control group, CSC, most closely matched the latency for the RPC subjects (refer figures 2-5, 2-6 and 2-7). Again, exposure to the apparatus for 1 hour on day three had little effect on conditioned fear reduction, in comparison to 1 hour RP treatment on the same day for other subjects. The CSC subjects had significantly longer latencies to first enter the FC compartment during sessions three and four in comparison to RPT and RPNT (refer table 2-20). Forced non-reinforced CS exposure is clearly an important factor in conditioned fear reduction.

Correlations Between the Dependent Variables

In order to assess if any relationship exists between the dependent variables, Pearson product-moment correlation coefficients were calculated across all subjects for each session. The results are presented in table 2-22. To further assess whether any relationship across subjects represents a between groups relationship, correlation coefficients were calculated using group means. The results are presented in table 2-23. It is clear from both sets of

TABLE 2-22

Pearson product-moment correlation coefficients between the dependent variables for all subjects across sessions.

| Correlation pair | | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| T-A[1] | correlation coefficient (r) | 0.046 | 0.437 | 0.473 | 0.491 |
| | probability level (p) | 0.360 | 0.000 | 0.000 | 0.000 |
| T-E[2] | r | -0.185 | 0.029 | -0.394 | -0.445 |
| | p | 0.073 | 0.412 | 0.001 | 0.000 |
| A-E[3] | r | -0.109 | 0.239 | -0.434 | -0.556 |
| | p | 0.197 | 0.029 | 0.000 | 0.000 |

1. Correlation between the time and approaches measures.
2. Correlation between the time and first entry latency measures.
3. Correlation between the approaches and first entry latency measure.

n = 63, df = 61, two-tailed test

TABLE 2-23

Pearson product-moment correlation coefficients between the dependent variables for group means across sessions.

| Correlation pair | | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| T-A[1] | Correlation coefficient (r) | -0.012 | 0.645 | 0.725 | 0.565 |
| | Probability level (p) | 0.488 | 0.030 | 0.013 | 0.057 |
| T-E[2] | r | -0.727 | -0.031 | -0.652 | -0.737 |
| | p | 0.013 | 0.468 | 0.028 | 0.012 |
| A-E[3] | r | -0.109 | 0.212 | -0.845 | -0.725 |
| | p | 0.390 | 0.292 | 0.002 | 0.014 |

1. Correlation between time and approaches measures.
2. Correlation between time and first entry latency measures.
3. Correlation between approaches and first entry latency measures.

n = 9, df = 7, two-tailed test

results that a positive relationship exists between the time and approach measures. On the other hand, a negative relationship exists between the latency measure and the other two dependent variables. As the latency to first enter the FC compartment increases there is a corresponding decrease in the time and approaches scores. This confirmed the pattern of results shown in figures 2-5, 2-6 and 2-7. Given these relationships, a two way (Groups x Sessions) multivariate analysis of variance was computed to assess the effects of the combined dependent variable scores.

Multivariate Analysis of Variance

With the use of Wilk's Lambda likelihood ratio criterion, the combined dependent variables were significantly affected by both group allocation, ($F(24,151) = 5.15$, $p<.001$) and sessions, ($F(9,389) = 36.53$, $p<.001$) and by their interaction, ($F(72,287) = 3.66$, $p<.001$).

The results indicated a high degree of association between group allocation and the combined dependent variables, $\eta^2 = 0.823$. A high degree of association was also found between sessions and the combined dependent variables, $\eta^2 = 0.774$.

As with experiment two, analyses of covariance were conducted to assess the effects of one dependent variable when the other dependent variables were treated as covariates, that is, held constant. In the first ANCOVA, the time measure acted as the dependent variable with the approaches and first entry latency measures acting as covariates. Time spent in the FC compartment varied across group allocation, ($F(8,52) = 4.36$, $p<.001$), across sessions ($F(3,160) = 49.14$, $p<.001$),

but was unaffected by their interaction ($F(24,160) = 1.25$, $p>.05$).

In the second ANCOVA, the approaches measure acted as the dependent variable, with the other two measures acting as covariates. Approaches into the FC compartment was significantly affected by group allocation, ($F(8,52) = 6.36$, $p<.001$), sessions ($F(3,160) = 15.83$, $p<.001$) and by their interaction ($F(24,160) = 3.84$, $p<.001$). In the third and final ANCOVA, the latency measure acted as the dependent variable with the other fear assessment measures acting as covariates. The latency to first enter the FC compartment was significantly affected by group allocation ($F(8,52) = 3.31$, $p<.005$), sessions ($F(3,160) = 25.98$, $p<.001$) and by their interaction ($F(24,160) = 3.45$, $p<.001$).

As with the results of the ANCOVAs performed in experiment two, the present ANCOVAs indicated that all dependent variables made unique contributions to the composite dependent variable used in the MANOVA analysis. It is clear that each dependent variable acted as a sensitive fear assessment measure.

To determine the relative contribution of the fear assessment measures to the multivariate dimension a stepwise discriminant function analysis was performed. A summary of the results is presented in table 2-24. The analysis yielded three discriminant functions, with a combined Chi Squared of ($X^2$ (24) = 130.49, $p<.001$). After removal of the first discriminent function, there still remained a highly significant amount of discriminating power, ($X^2$ (14) = 54.541, $p<.001$). After the removal of the second discriminant function, there remained an insignificant amount of

TABLE 2-24

(a) Canonical Discriminant Functions - Summary Table

| Function | Eigenvalue | % of Variance | Canonical Correlation | After Function | Wilks Lambda | $X^2$ | d.f. | Significance |
|---|---|---|---|---|---|---|---|---|
| 1 | 0.363 | 60.23 | 0.516 | 0 | 0.587 | 130.490 | 24 | 0.000 |
| 2 | 0.191 | 31.66 | 0.401 | 1 | 0.800 | 54.541 | 14 | 0.000 |
| 3 | 0.049 | 8.11 | 0.216 | 2 | 0.953 | 11.701 | 6 | 0.069 |

(b) Loading Matrix between predictor variables and discriminant functions.

| Measure | Function 1 | Function 2 |
|---|---|---|
| Time | -0.809 | 0.111 |
| Approaches | -0.284 | -0.753 |
| First entry latency | 0.663 | 0.510 |

(c) Standardised Canonical Discriminant Function Coefficients

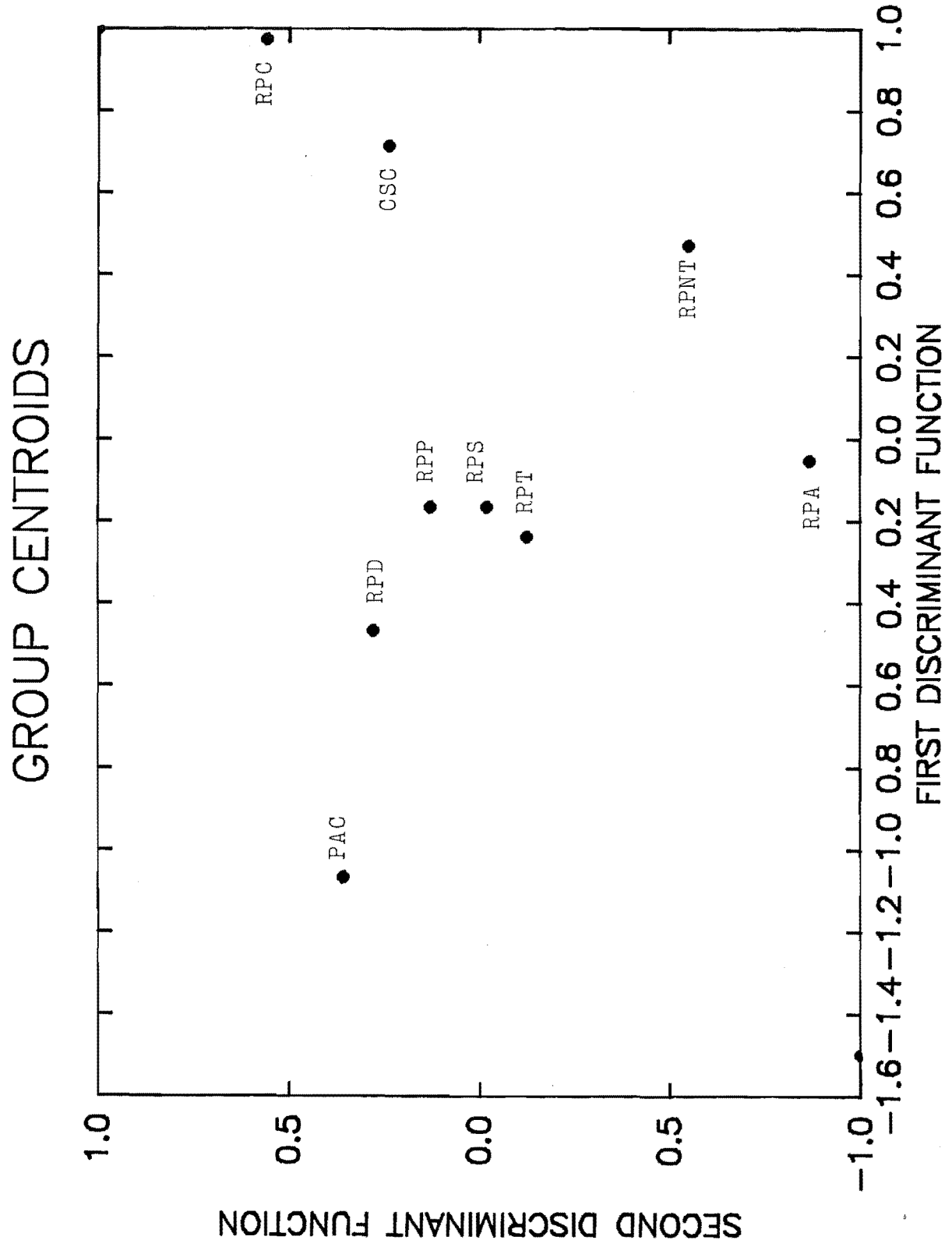
| Measure | Function 1 | Function 2 |
|---|---|---|
| Time | -0.846 | 0.650 |
| Approaches | 0.276 | -0.977 |
| First entry latency | 0.593 | 0.374 |

discriminating power, ( $\chi^2$ (6) = 11.701, p>.05), therefore the third discriminant function was dropped from further consideration. The first two discriminant functions accounted for 60.23% and 31.66%, respectively, of the between-groups variability, (see table 2-24(a)). Thus, the groups differed in at least two significant ways.

The canonical discriminant functions evaluated at group means (group centroids) are plotted in figure 2-8. As illustrated in figure 2-8, the first discriminant function maximally separates RPC, CSC and RPNT groups from the PAC subjects, with the remaining groups, RPA, RPT, RPS, RPP and RPd forming a cluster between RPC and PAC groups. The groups forming the cluster, or to put it another way, which are indistinguishable on the first discriminant function, all had therapist assisted RP treatment, some with drugs as adjuncts to RP, RPD, RPP and RPA, while RPS and RPT were without drug treatments. It is clear that the first discriminant function has discriminated between firstly, therapist assisted RP treatment and non therapist RP treatment and secondly, between the different control procedures, PAC vs CSC and RPC groups. The second discriminant function maximally separates RPC and RPA groups, with PAC, RPd and CSC groups being indistinguishable from each other, with RPP, RPS and RPT groups falling in between the maximally separated groups. The RPNT subjects most closely resembled the RPA group on the second discriminant function.

A loading matrix between the measures and discriminant functions, (see table 2-24(b)), indicates that the first discriminant function is correlated most highly with the time measure (r= -0.809), while the second discriminant function

FIGURE 2-8 Group centroids in the discriminant space formed by the first and second discriminant functions.



is correlated most highly with the approaches measure (r= -0.753). The first entry latency measure is moderately correlated with both discriminant functions, r = 0.663, and r = 0.510, respectively. The approaches measure has only a low correlation with the first discriminant function (r = -0.284) as does the time measure with the second discriminant function (r = 0.111). This suggests that the maximum spread among the groups on the first discriminant function is primarily based on the time measure scores, with the greatest difference obtained between RPC and PAC groups, as expected, (refer to figure 2-8). It also suggests that the maximum spread among the groups on the second discriminant function is primarily based on the approaches measure scores, with the greatest difference obtained between RPC and RPA groups. This is not surprising given the relatively high number of approaches performed by the RPA subjects (cf. figure 2-5). The first entry latency scores made a moderate contribution to group separation on both discriminant functions, thus, the first entry latency measure had a mediating influence on both the time and approaches measures. It is of particular theoretical interest to note that while RPC and PAC control groups are maximally discriminated on the first discriminant function, the time measure, 0.974 and -1.067, respectively, these two groups are indistinguishable on the second discriminant function, the approaches measure, -0.128 and -0.090, respectively. The implication of this result for the fear assessment measures will be discussed in the general discussion section at the end of this chapter.

Examination of the results of the stepping procedure used to enter the measures into the discriminant function

analysis indicated that the time measure had the highest discriminating power of the measures as it entered the analysis first, followed by the first entry latency measure and finally by the approaches measure. All measures produced significant changes in Rao's V when included in the analysis; for the time measure (change in Rao's V = 62.40, $p<.001$), for the first entry latency measure (change in Rao's V = 43.89, $p<.001$), and for the approaches measure (change in Rao's V = 40.34, $p<.001$). The inclusion of the first entry latency measure into the analysis before the approaches measure probably reflects the moderate correlation of the first entry measure with both canonical discriminant functions producing a slightly greater discriminating power in comparison to the approaches measure. But it is also related to the criteria chosen for inclusion into the analysis, namely, Rao's V. Another stepwise discriminant function analysis was conducted with the criteria for entry into the analysis being the size of Wilk's Lambda, or the overall multivariate F ratio for the test of differences among the group centroids. In this analysis the time measure was entered first, followed by the approaches measure, and finally the first entry latency entered the analysis. Clearly, the criteria chosen for the inclusion of variables into the analysis effects the outcome of the analysis, therefore, consideration of a number of statistics is required when interpreting the results of a discriminant function analysis.

In summary, the above analyses indicated that all three measures made significant contributions to the discriminant function analysis. The time measure correlated most highly

with the first discriminant function while the approaches measure correlated the highest with the second discriminant function. The first entry latency exerted its influence on both discriminant functions, but only in a secondary manner in comparison to the other measures. Overall, the analyses indicated that the time measure had a higher discriminating power relative to the other measures which, in turn, exerted their lesser discriminating power in different ways. The most sensitive fear measure in discriminating the RP treatment groups from the control groups was the time measure, with some assistance from the first entry latency measure. The approaches measure was primarily correlated with the second discriminant function which surprisingly failed to discriminate between the control groups, RPC and PAC, yet did discriminate between the various RP treatment groups. It would seem that the approaches measure is more sensitive to the processes involved in RP treatment and conditioned fear and avoidance extinction than the processes involved in conditioned fear and avoidance maintenance. This will be discussed further at the end of this chapter.

The results of the present experiment further extended and corroborated the findings reported in experiment two. The time, approaches and latency measures were all found to be sensitive to conditioned fear acquisition, RP treatment and conditioned fear reduction. That each dependent variable was sensitive to different aspects of conditioned fear was illustrated with respect to the social facilitation effect. The social facilitation effect was obtained when measured by time spent in the FC compartment, partially obtained when

assessed by the latency to first enter the FC compartment, and not obtained when assessed by approaches into the FC compartment. This finding indicated a gradient of sensitivity extisted across the dependent variables with respect to the social facilitation effect. The question of what particular aspects and processes of conditioned fear the individual dependent variables were sensitive to, was not addressed by the current study, because it was beyond the scope of the study. But it is of immense interest that one method used to increase the efficacy of RP treatment, social facilitation, can be confirmed using one fear assessment method and not confirmed using another. By examining a number of measures used to increase the efficacy of RP treatment within the present experiment's methodological framework a behavioural profile of the dependent variables could be drawn thus providing some answers to the issue raised above.

The use of drugs as adjuncts to RP treatment produced disappointing results. When assessed by the time dependent variable, only the beta-adrenergic blocker, propranolol, significantly facilitated the effects of RP treatment in comparison to saline controls. But rather surprisingly, this effect, present during session three, disappeared during session four (refer figure 2-5 and table 2-14). The beta-adrenergic blocker, atenolol, and the benzodiazepine, diazepam, had no effect on increasing the efficacy of RP treatment. When the drug assisted RP treatment effects were assessed using the approaches dependent variable, only atenolol significantly increased the efficacy of RP treatment in reducing conditioned fear and avoidance of the FC compartment. This interpretation must be treated with some caution as RPA

subjects showed a high overall level of approaches into the FC compartment, with RPA subjects significantly entering the FC compartment more times during session one than RPS, RPd, RPA and RPC subjects. It is possible that RPA subjects had an overall higher level of general motor activity in comparison to RPS subjects which produced the atenolol effect, rather than the effect being an atenolol facilitated RP treatment effect per se. Propranolol assisted RP treatment subjects performed more approaches into the FC compartment in comparison to saline controls (refer figure 2-5), but this difference was non significant. There were no significant differences in the latency measure among RPS, RPd, RPP and RPA subjects, except for the result that RPP subjects increased their latency to first enter the FC compartment during session four in comparison to saline controls (refer to figure 2-7). This also had the effect of contributing to the decrease in the time measure during session four by RPP subjects. This interpretation is confirmed by the correlational analyses reported in tables 2-22 and 2-23.

Diazepam assisted RP treatment consistently failed to produce a facilitative RP treatment effect across all dependent variables. These subjects most closely resembled the saline controls. While propranolol, atenolol and diazepam failed to consistently produce a facilitative RP treatment effect, never-the-less, a RP treatment effect was consistently obtained with these groups across all dependent variables.

In the present experiment each drug was assessed by a single dose level, and thus it was impossible to obtain a dose-response function for each drug. It is well known that

a drug's behavioural effect is a function of a number of factors, one of which is dose level (Carlton, 1983). Given this, it is not known whether the dose levels chosen for the present experiment were optimal to obtain the behavioural effects under investigation. The following experiment was therefore conducted to assess the dose-response function of propranolol assisted RP treatment using the same approach assessment measures of the present experiment.

# EXPERIMENT FOUR

## Drug Assisted Response Prevention Treatment: A Dose-response Analysis for Propranolol.

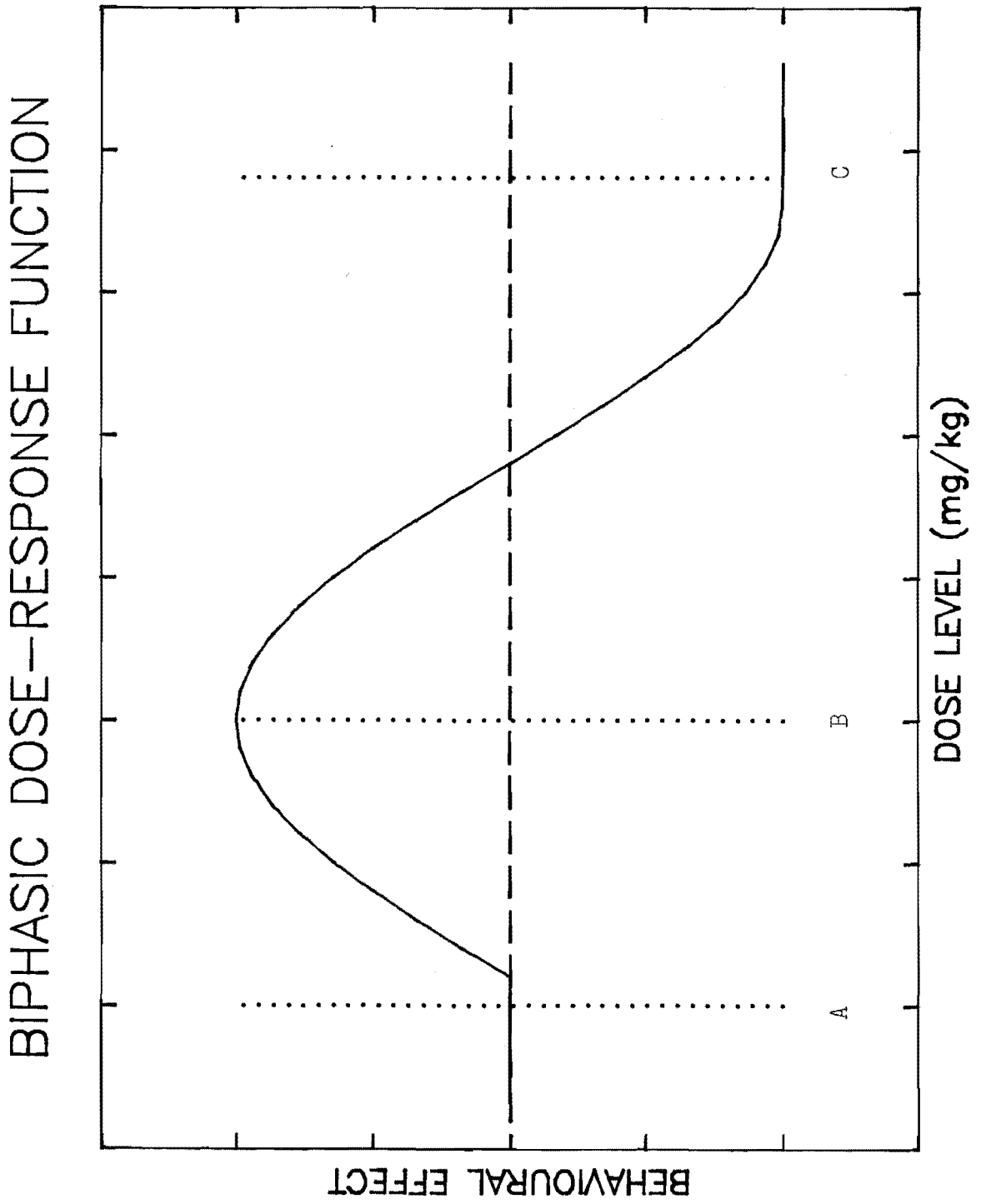
### Introduction

In experiment three, each drug was examined at a single moderate dose level, with saline controls acting as a zero dose for each drug. Yet it is known that different dosages of a particular drug produce different behavioural effects. One possible dose-response function has a biphasic behavioural effect. This is illustrated in figure 2-9.

While not used in figure 2-9, a logarithmic transformation of the X-axis (dose level) is often employed to illustrate a dose-response function. This permits a large range of dosages to be displayed in a single graph and allows comparison between different dose-response functions.

At point A on figure 2-9, a low dose of the drug is no different in behavioural effect from a control dosage, which might lead the researcher to interpret the finding as indicating that the drug is biologically inactive. Yet if the researcher had chosen a dose level at B on figure 2-9 an entirely different interpretation would be required. At B, the dosage has produced an increase in behavioural activity in comparison to the control dosage, termed a stimulant effect. Also point B on this dose-response function is the dose level with maximum behavioural effect. The steeper the slope of the function the smaller the increase in dose level required to move from a minimal to maximal behavioural effect. However, it is important to remember that doubling the dosage does not necessarily produce double the behavioural

FIGURE 2-9 A biphasic dose-response function.



effect.

Finally, at point C on figure 2-9, the drug dose level has produced decreased behavioural effect in comparison to the control dosage, termed a depressant effect. Clearly, to adequately characterize a drug's action on behavioural activity more than one dose level is required.

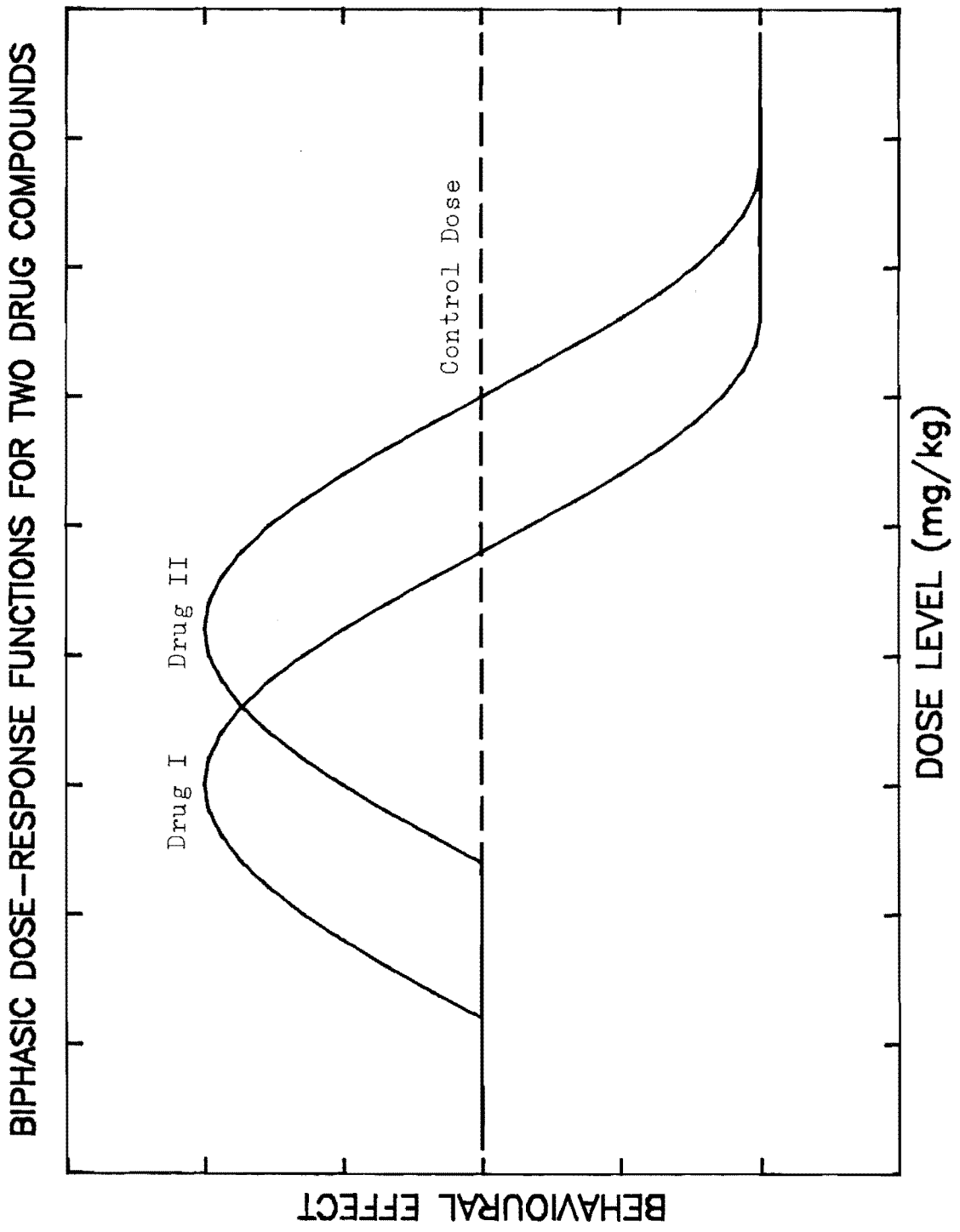
While figure 2-9 illustrates a biphasic dose-response function, monophasic dose-response functions are also observed. That is, increasing the drug's dose level produces one effect only on behavioural activity. An example of a monophasic dose-response function is provided by neuroleptic drugs. They decrease avoidance and escape responding at a rate directly proportional to the dose level.

It is therefore clear that experiment three suffers from a methodological weakness in respect of the drug analysis. The interpretation of drug effects was based on only a moderate dose level for each drug, which limits comparisons between drugs. This is illustrated in figure 2-10.

It can be seen that drug I produces both increases and decreases in behavioural activity at lower dose levels in comparison to drug II. This indicated drug I is relatively more potent than drug II, with drug I being quantitatively different from drug II, but not qualitatively. However, if the drugs are examined at various equivalent dose levels along the X-axis in isolation, one might conclude that there are qualitative differences between the drugs when they do not exist.

It is possible that the dose level with the maximum behavioural effect (point B on figure 2-9) was not chosen in experiment three.

FIGURE 2-10 Biphasic dose-response functions for two drug compounds.



The purpose of the present experiment is to examine a range of dose levels for the non-selective beta-adrenergic blocker, propranolol, which will provide a more adequate characterization of propranolol assisted RP treatment in reducing conditioned fear and avoidance.

## Method

### Subjects

The subjects were 28 male New Zealand random bred Wistar rats. At the time of testing the subjects had a mean age of 171 days with a mean weight of 317 grams. The maintenance schedule was identical to that of experiment one.

### Apparatus

The apparatus was identical to that of experiment three.

### Procedure

Prior to the beginning of the experiment, the subjects were randomly allocated to one of four groups (n = 7). One group of subjects was assigned to the 5.0 mg/kg propranolol condition (RPP5), another was assigned to the 15.0 mg/kg propranolol condition, (RPP15) with the remaining subjects being assigned to one of the two groups of therapist rats. The experimental procedure was divided into four phases according to the sequence of events described for experiment three.

## Results and Discussion

In addition to groups RPP5 and RPP15, the data from the following groups of experiment three were included for purposes

of comparative analysis, RPP, RPS, RPC and PAC. Group RPP from experiment three was injected with 10 mg/kg of propranolol prior to RP treatment and is included as another dose-level. Group RPS was included as a drug control group. The inclusion of these groups produces four dose levels for propranolol, (0 mg/kg (RPS), 5 mg/kg (RPP5), 10 mg/kg (RPP10) and 15 mg/kg (RPP15). The response prevention control group, RPC, and the fear conditioning control group, PAC, were included to allow assessment of RP treatment and fear conditioning. The raw scores yielded by the dependent variables were treated in an identical fashion to that reported in experiment three.

Time Spent in the FC Compartment

Group means and standard deviations across sessions are presented in table 2-25, with the means being graphically represented in figure 2-11.

A split-plot ANOVA revealed that time spent in the FC compartment was affected by both group membership, ($F(5,36) = 8.67$, $p<.001$), and session number, ($F(3,108) = 20.77$, $p<.001$), and by the interaction of group membership and session number, ($F(15,108) = 7.20$, $p<.001$). As reported in experiments two and three, all subjects again spent an equivalent amount of the time in the FC compartment during habituation, session one, ($F(5,36) = 1.30$, $p>.05$), although there was a slight bias in favour of the other compartment, with the subjects spending an average 42.5% of the session time in the FC compartment.

The effects of the fear conditioning procedure during session two is illustrated in figure 2-11. Tukey multiple comparison tests, summarized in table 2-26, confirmed that

TABLE 2-25

Means and standard deviations of proportional time spent in the FC compartment.

| Treatment Group | | Sessions | | | | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | Overall |
| PAC | M | 0.443 | 0.519 | 0.507 | 0.489 | 0.489 |
| | SD | 0.080 | 0.092 | 0.052 | 0.105 | 0.085 |
| RPC | M | 0.434 | 0.149 | 0.004 | 0.004 | 0.148 |
| | SD | 0.061 | 0.030 | 0.005 | 0.008 | 0.182 |
| RPS | M | 0.430 | 0.187 | 0.397 | 0.424 | 0.360 |
| | SD | 0.069 | 0.054 | 0.270 | 0.186 | 0.190 |
| RPP5 | M | 0.397 | 0.116 | 0.546 | 0.233 | 0.323 |
| | SD | 0.032 | 0.050 | 0.280 | 0.150 | 0.225 |
| RPP10 | M | 0.383 | 0.130 | 0.766 | 0.327 | 0.401 |
| | SD | 0.091 | 0.024 | 0.292 | 0.304 | 0.311 |
| RPP15 | M | 0.461 | 0.164 | 0.407 | 0.469 | 0.375 |
| | SD | 0.057 | 0.028 | 0.325 | 0.324 | 0.252 |
| Overall | M | 0.425 | 0.211 | 0.438 | 0.324 | |
| | SD | 0.069 | 0.149 | 0.323 | 0.260 | |

FIGURE 2-11 Proportional time spent in the FC compartment for each group across sessions.

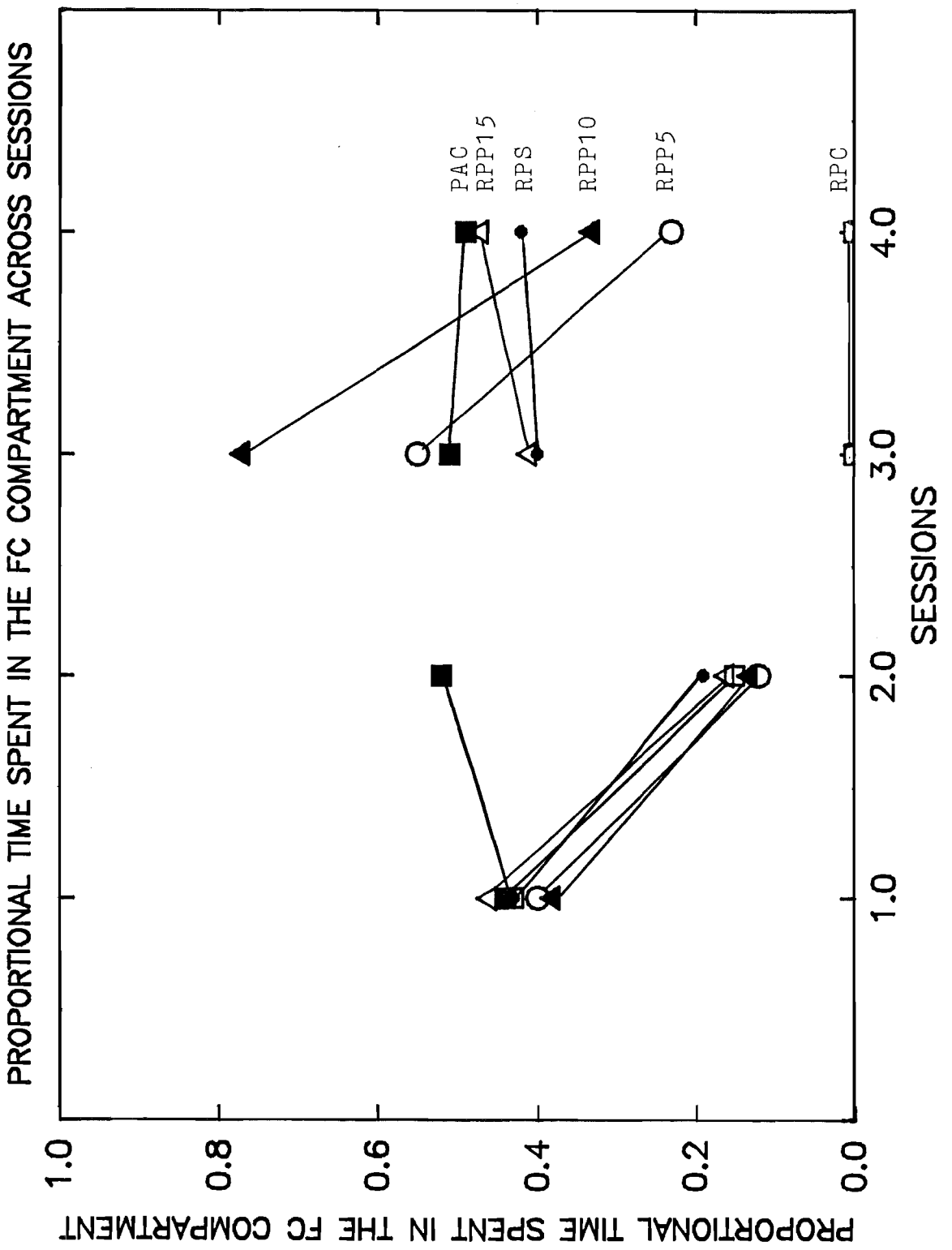


TABLE 2-26

Summary of the Tukey comparison tests for the Groups Factor at each level of the Sessions Factor : Time scores.

| Group Comparisons | Sessions[1] 2 | 3 | 4 |
|---|---|---|---|
| RPC-PAC | 5.968 | 8.113 | 7.823 |
| RPC-RPS | NS[2] | 6.339 | 6.774 |
| RPC-RPP5 | NS | 8.742 | 3.694 |
| RPC-RPP10 | NS | 12.290 | 5.210 |
| RPC-RPP15 | NS | 6.500 | 7.500 |
| PAC-RPS | 5.355 | NS | NS |
| PAC-RPP5 | 6.500 | NS | 4.129 |
| PAC-RPP10 | 6.274 | 4.177 | NS |
| PAC-RPP15 | 5.726 | NS | NS |
| RPS-RPP5 | NS | NS | 3.081 |
| RPS-RPP10 | NS | 5.952 | NS |
| RPS-RPP15 | NS | NS | NS |
| RPP5-RPP10 | NS | 3.548 | NS |
| RPP5-RPP15 | NS | NS | 3.806 |
| RPP10-RPP15 | NS | 5.790 | NS |

1. session one results were not included as they are all non-significant
2. non-significant comparison $q'.05 = 2.837$, $q'.01 = 3.769$

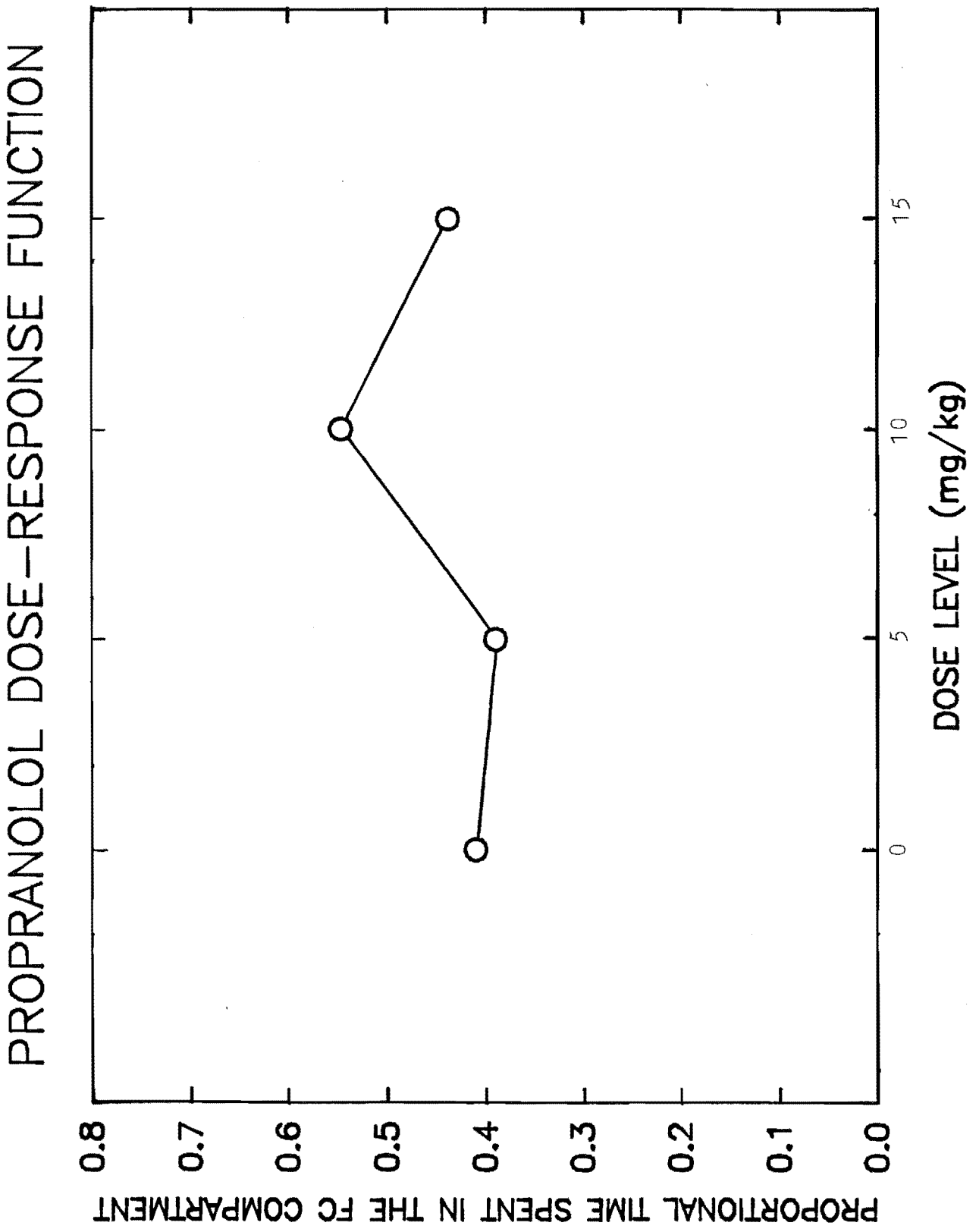
all groups significantly differed from PAC subjects. This difference was due to fear conditioning significantly reducing the time spent in the FC compartment. That this reduction was constant for all groups was confirmed by Tukey tests (see table 2-26). Only PAC subjects increased their time spent in the FC compartment during session two.

The time measure varied between groups during both session three, ($F(5,36) = 7.66$, $p<.001$), and session four, ($F(5,36) = 5.33$, $p<.001$). Tukey comparison tests confirmed that this variation was primarily due to RPC subjects spending significantly less time in the FC compartment in comparison to RP treatment groups (refer to table 2-12). Clearly, RP treatment has eliminated the effects of fear conditioning.

Of particular interest in the present experiment is the relationship between the time measure and the dose level of propranolol assisted RP treatment. A dose-response function for propranolol is presented in figure 2-12, with the mean time spent in the FC compartment collapsed over sessions three and four plotted against propranolol dose-level. Although both 10.0 mg/kg and 15.0 mg/kg of propranolol increased the efficacy of RP treatment in comparison to saline controls, the increase failed to reach statistical significance across extinction sessions (refer to table 2-26). The 5.0 mg/kg dosage of propranolol had no effect on the proportional time spent in the FC compartment. Examination of figure 2-12 indicates that within the range tested the optimal dose-level of propranolol for increasing the efficacy of RP treatment is 10.0 mg/kg, the dose-level selected in experiment three.

FIGURE 2-12 Propranolol dose-response function :

Proportional time scores as a function of dose level.



Approaches into the FC Compartment

Group means and standard deviations across groups are presented in table 2-27, with the means being graphically represented in figure 2-13.

A split-plot ANOVA revealed that approaches into the FC compartment were affected by both group membership, ($F(5,36) = 7.06$, $p<.001$), and session number ($F(3,108) = 21.72$, $p<.001$), and by their interaction, ($F(15,108) = 2.35$, $p<.01$).

Approaches into the FC compartment during session one significantly varied across groups, ($F(5,36) = 3.25$, $p<.02$). Subsequent Tukey comparison tests, summarized in table 2-28, confirmed that this variation was due to the high number of approaches performed by RPP15 subjects in comparison to the low number of approaches performed by the control subjects, PAC and RPC. All other comparisons were non-significant. The effect of fear conditioning during session two is illustrated in figure 2-13. Tukey multiple comparison tests, summarized in table 2-29, confirmed that all RP treatment groups significantly reduced the number of approaches into the FC compartment.

The number of approaches into the FC compartment varied between groups during session three, ($F(5,36) = 7.11$, $p<.001$), and session four ($F(5,36) = 3.26$, $p<.02$). Again, Tukey comparison tests confirmed that this variation was primarily due to RPC subjects significantly reducing the number of approaches into the FC compartment in comparison to RP treatment groups (refer to table 2-28).

The relationship between the approaches measure and the

TABLE 2-27

Means and standard deviations of approaches per minute into the FC compartment.

| Treatment Group | | Sessions 1 | 2 | 3 | 4 | Overall |
|---|---|---|---|---|---|---|
| PAC | M | 2.400 | 1.989 | 1.287 | 1.274 | 1.738 |
| | SD | 0.802 | 0.685 | 0.484 | 0.445 | 0.763 |
| RPC | M | 2.230 | 1.297 | 0.076 | 0.197 | 0.950 |
| | SD | 0.759 | 0.283 | 0.099 | 0.349 | 0.988 |
| RPS | M | 2.786 | 1.494 | 1.651 | 1.757 | 1.922 |
| | SD | 0.905 | 0.716 | 0.581 | 0.646 | 0.855 |
| RPP5 | M | 3.251 | 1.727 | 3.231 | 2.616 | 2.706 |
| | SD | 0.392 | 1.066 | 1.922 | 1.535 | 1.425 |
| RPP10 | M | 2.817 | 1.593 | 2.123 | 1.486 | 2.005 |
| | SD | 0.374 | 0.525 | 1.115 | 1.144 | 0.974 |
| RPP15 | M | 3.583 | 1.729 | 1.390 | 2.171 | 2.218 |
| | SD | 0.999 | 0.262 | 0.926 | 2.129 | 1.469 |
| Overall | M | 2.845 | 1.638 | 1.626 | 1.584 | |
| | SD | 0.841 | 0.648 | 1.362 | 1.378 | |

FIGURE 2-13 Approaches per minute into the FC compartment for each group across sessions.

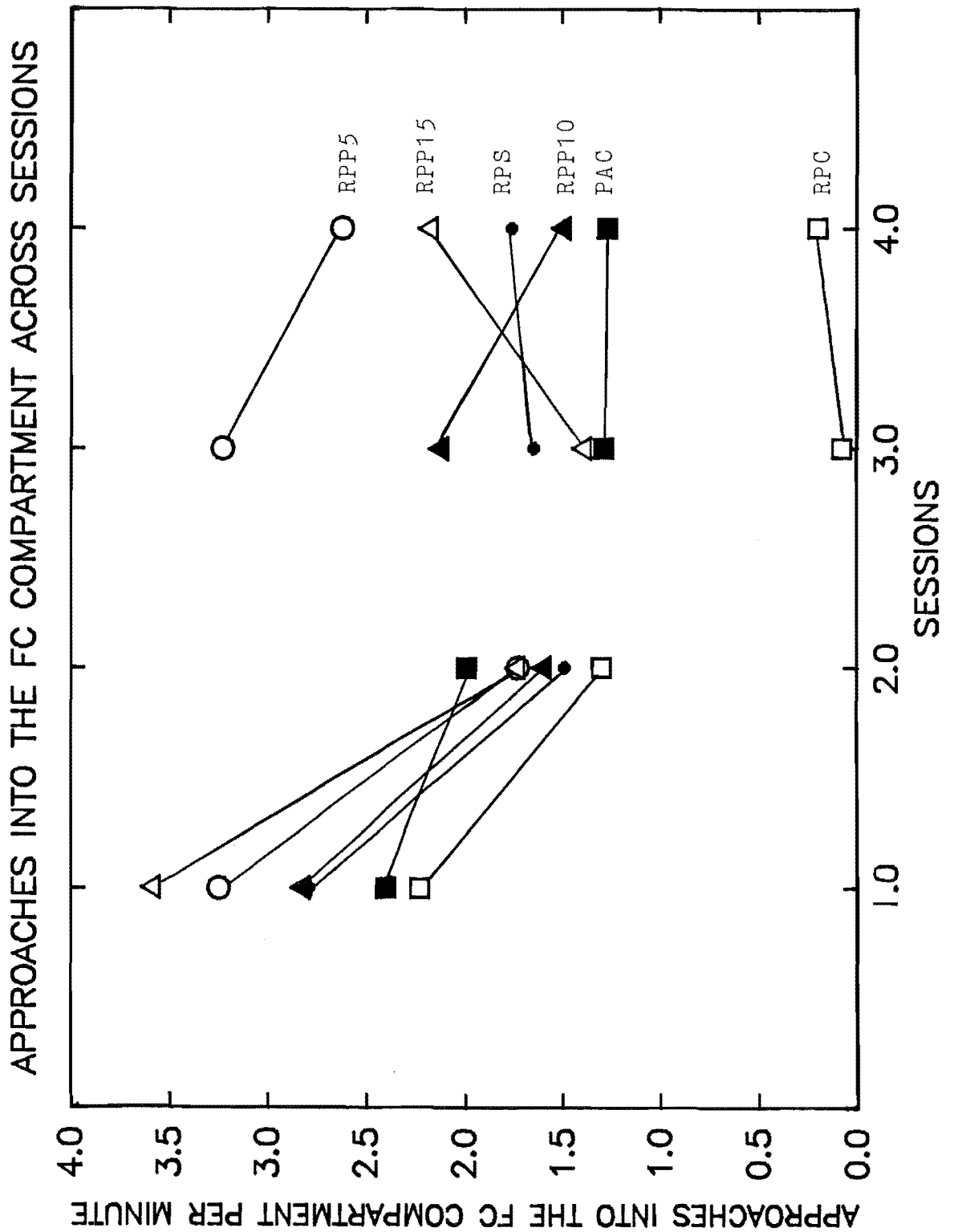


TABLE 2-28

Summary of the Tukey comparison tests for the Groups Factor at each level of the Sessions Factor : Approaches into the FC compartment scores.

| Group Comparisons | Sessions[1] 1 | 3 | 4 |
|---|---|---|---|
| RPC-PAC | NS[2] | 3.411 | 3.034 |
| RPC-RPS | NS | 4.437 | 4.394 |
| RPC-RPP5 | 2.876 | 8.887 | 6.814 |
| RPC-RPP10 | NS | 5.766 | 3.631 |
| RPC-RPP15 | 3.811 | 3.701 | 5.561 |
| PAC-RPS | NS | NS | NS |
| PAC-RPP5 | NS | 5.476 | 3.780 |
| PAC-RPP10 | NS | NS | NS |
| PAC-RPP15 | 3.332 | NS | NS |
| RPS-RPP5 | NS | 4.451 | NS |
| RPS-RPP10 | NS | NS | NS |
| RPS-RPP15 | NS | NS | NS |
| RPP5-RPP10 | NS | 3.121 | 3.183 |
| RPP5-RPP15 | NS | 5.186 | NS |
| RPP10-RPP15 | NS | NS | NS |

1. session two results were not included as they are all non-significant
2. non-significant comparison $q'.05 = 2.836$ $q'.01 = 3.769$

TABLE 2-29

Summary of the Tukey comparison tests for the Sessions Factor at each level of the Groups Factor : Approaches into the FC compartment scores.

| Treatment Group | Session comparisons | | | | | |
|---|---|---|---|---|---|---|
| | 1-2 | 1-3 | 1-4 | 2-3 | 2-4 | 3-4 |
| PAC | NS[1] | NS | NS | NS | NS | NS |
| RPC | NS | 6.669 | 6.294 | 3.780 | NS | NS |
| RPS | 4.00 | NS | NS | NS | NS | NS |
| RPP5 | 4.718 | NS | NS | 4.656 | NS | NS |
| RPP10 | 3.789 | NS | 4.121 | NS | NS | NS |
| RPP15 | 5.740 | 6.789 | 4.371 | NS | NS | NS |

df = 4, 108

q.05 = 3.70

q.01 = 4.52

1 = non-significant comparison

dose level of propranolol is illustrated in figure 2-14. The number of approaches into the FC compartment collapsed over sessions three and four and is plotted against dose level. Only 5.0 mg/kg propranolol assisted RP treatment increased the number of approaches into the FC compartment in comparison to saline controls (o mg/kg dose level). Thus, the optimal dose level of propranolol for increasing the efficacy of RP treatment is 5.0 mg/kg, when assessment is by the approaches measure. When fear assessment is time spent in the FC compartment, the optimal dose-level is 10.0 mg/kg. This result again raises the possibility that the fear assessment measures are sensitive to different aspects of conditioned fear. Given this, it is not surprising to obtain different dose-response functions for propranolol for the fear assessment measures.

Latency to First Enter the FC Compartment

Group means and standard deviations across groups are presented in table 2-30, with the means being grapically represented in figure 2-15.

A split-plot ANOVA revealed that the log latency to first enter the FC compartment was significantly affected by both group membership, ($F(5,36) = 7.16$, $p<.001$), and session number ($F(3,108) = 10.81$, $p<.001$) and by their interaction, ($F(15,108) = 4.81$, $p<.001$).

As expected, there was no significant variation across groups in the latency measure during session one, ($F(5,36) = 0.92$, $p>.05$), and session two, ($F(5,36) = 1.31$, $p>.05$), but the log latency to first enter the FC compartment significantly varied across groups during session three, ($F(5,36) = 6.69$,

FIGURE 2-14 Propranolol dose-response function : Approach scores as a function of dose level.

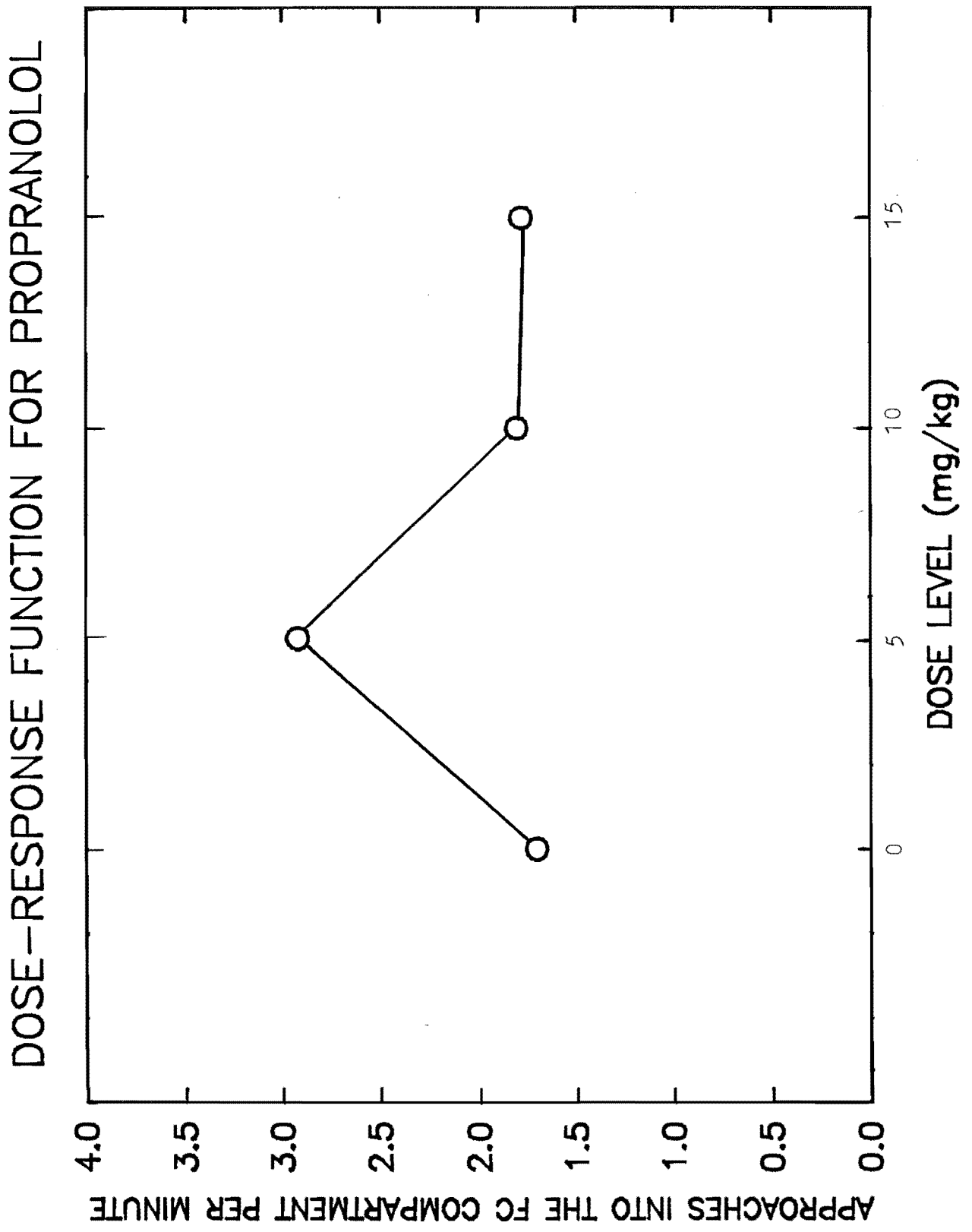
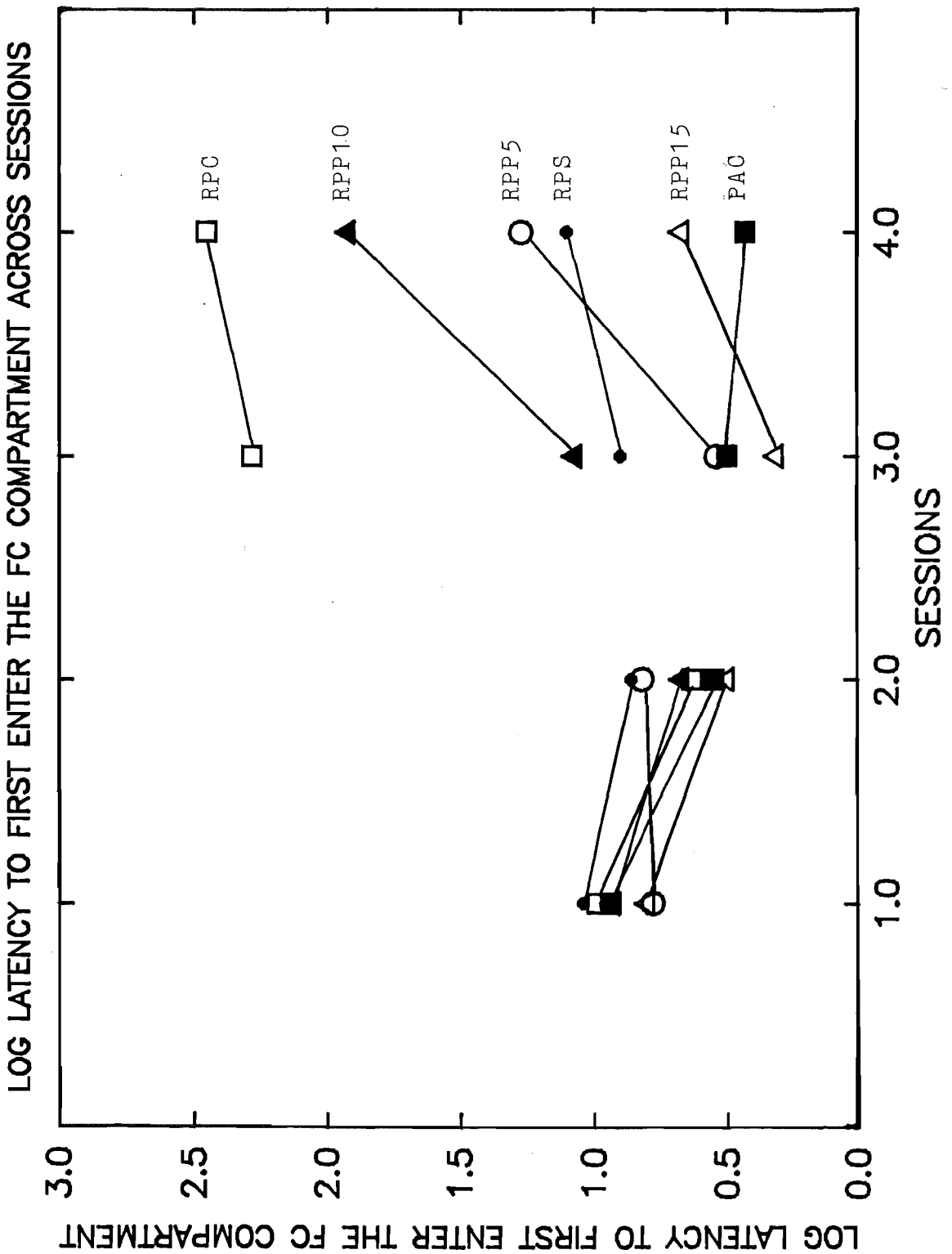


TABLE 2-30

Means and standard deviations of the log latency to first enter the FC compartment.

| Treatment Group | | Sessions | | | | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | Overall |
| PAC | M | 0.937 | 0.551 | 0.497 | 0.426 | 0.603 |
| | SD | 0.243 | 0.196 | 0.254 | 0.242 | 0.299 |
| RPC | M | 0.994 | 0.633 | 2.279 | 2.447 | 1.588 |
| | SD | 0.128 | 0.231 | 1.260 | 0.735 | 1.064 |
| RPS | M | 1.036 | 0.863 | 0.899 | 1.101 | 0.974 |
| | SD | 0.266 | 0.235 | 0.374 | 0.635 | 0.398 |
| RPP5 | M | 0.779 | 0.816 | 0.544 | 1.266 | 0.851 |
| | SD | 0.352 | 0.357 | 0.666 | 0.983 | 0.663 |
| RPP10 | M | 0.927 | 0.674 | 1.067 | 1.919 | 1.147 |
| | SD | 0.277 | 0.515 | 0.962 | 1.232 | 0.919 |
| RPP15 | M | 0.799 | 0.500 | 0.309 | 0.671 | 0.569 |
| | SD | 0.377 | 0.344 | 0.236 | 0.547 | 0.415 |
| Overall | M | 0.912 | 0.673 | 0.932 | 1.305 | |
| | SD | 0.284 | 0.337 | 0.953 | 1.024 | |

FIGURE 2-15 Log latency to first enter the FC compartment for each group across sessions.



$p<.001$), and session four, ($F(5,36) = 6.43$, $p<.001$). The Tukey multiple comparison tests, summarized in table 2-31, confirmed that the RPC subjects took significantly longer to first enter the FC compartment in comparison to RP treatment groups. Response prevention groups significantly reduced conditioned fear and avoidance of the FC compartment.

The relationship between the latency measure and the dose-level of propranolol is presented in figure 2-16. The only dose-level of propranolol to reduce the latency to first enter the FC compartment was 15.0 mg/kg. The 10.0 mg/kg dose increased the latency while the 5.0 mg/kg dose had no effect. Thus, the optimal dose of propranolol to increase the efficacy of RP treatment, as measured by the latency measure, is 15.0 mg/kg. Each fear assessment measure has produced its own characteristic dose-response function for propranolol, raising the possibility that the fear assessment procedures are measuring behaviours that are mediated by different processes.

Correlations Between the Fear Assessment Measures

In order to assess if any relationship exists between the measures, Pearson product-moment correlation coefficients were calculated across all subjects for each session. The results are presented in table 2-32. To further assess whether any relationship across subjects represents a between groups relationship, correlation coefficients were calculated using group means. The results are presented in table 2-33. Once again, a positive relationship exists between the time and approaches measures. A negative relationship exists between the approaches and first entry latency measures.

TABLE 2-31

Summary of the Tukey comparison tests for the Groups Factor at each level of the Sessions Factor : Log latency to first enter the FC compartment scores.

| Group Comparisons | Sessions[1] 3 | 4 |
|---|---|---|
| RPC-PAC | 8.100 | 9.186 |
| RPC-RPS | 6.273 | 6.118 |
| RPC-RPP5 | 7.886 | 5.368 |
| RPC-RPP10 | 5.509 | NS |
| RPC-RPP15 | 8.954 | 8.073 |
| PAC-RPS | NS[2] | 3.068 |
| PAC-RPP5 | NS | 3.818 |
| PAC-RPP10 | NS | 6.786 |
| PAC-RPP15 | NS | NS |
| RPS-RPP5 | NS | NS |
| RPS-RPP10 | NS | 3.718 |
| RPS-RPP15 | NS | NS |
| RPP5-RPP10 | NS | 2.968 |
| RPP5-RPP15 | NS | NS |
| RPP10-RPP15 | NS | 5.673 |

1. Sessions 1 and 2 results were not included as they were all non-significant.
2. Non-significant comparison $q'.05 = 2.839$, $q'.01 = 3.774$

FIGURE 2-16 Propranolol dose-response function : Latency scores as a function of dose level

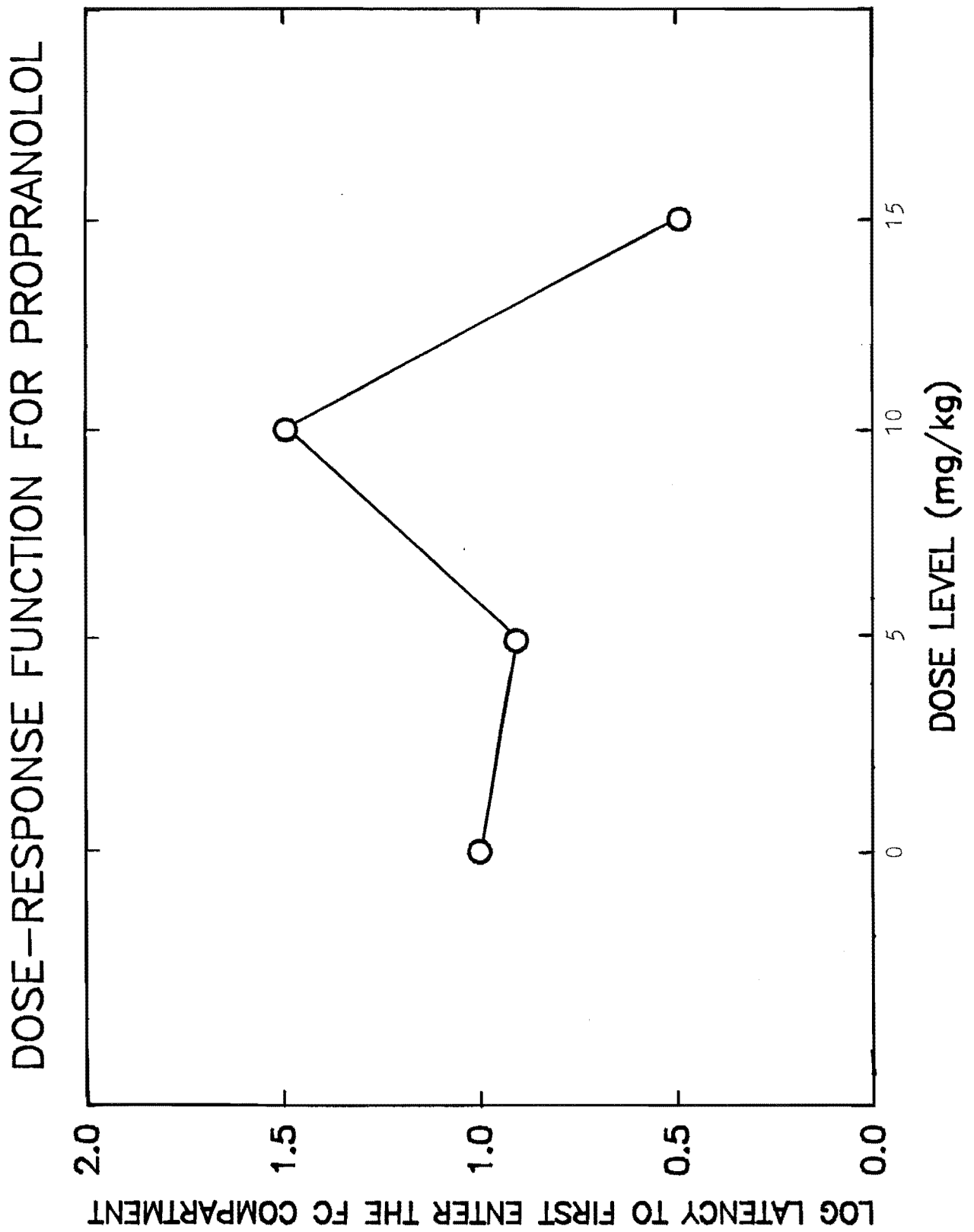


TABLE 2-32

Pearson product-moment correlation coefficients between the fear assessment measures for all subjects across sessions.

| Correlation pair | | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| T-A[1] | Correlation coefficient (r) | 0.092 | 0.297 | 0.300 | 0.378 |
| | Probability level (p) | 0.281 | 0.028 | 0.027 | 0.007 |
| T-E[2] | r | 0.064 | -0.179 | -0.364 | -0.544 |
| | p | 0.343 | 0.129 | 0.009 | 0.000 |
| A-E[3] | r | -0.236 | -0.179 | -0.372 | -0.384 |
| | p | 0.066 | 0.128 | 0.008 | 0.006 |

1. Correlation between the time and approaches measures.
2. Correlation between the time and first entry latency measures.
3. Correlation between the approaches and first entry latency measures.

n = 42, df = 4, two-tailed test

TABLE 2-33

Pearson product-moment correlation coefficients between the fear assessment measures for group means across sessions.

| Correlation pair | | Sessions 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| T-A[1] | Correlation coefficient (r) | 0.092 | 0.729 | 0.762 | 0.519 |
| | Probability level (p) | 0.431 | 0.050 | 0.039 | 0.146 |
| T-E[2] | r | -0.002 | -0.339 | -0.661 | -0.880 |
| | p | 0.498 | 0.255 | 0.076 | 0.010 |
| A-E[3] | r | -0.793 | -0.345 | -0.634 | -0.571 |
| | p | 0.030 | 0.251 | 0.088 | 0.118 |

1. Correlation between time and approaches measures.
2. Correlation between time and first entry latency measures.
3. Correlation between approaches and first entry latency measures.

n = 6, df = 4, two-tailed test

Given these relationships, a two-way, Groups by Sessions, multivariate analysis of variance, MANOVA, was performed.

Multivariate Analysis of Variance

With the use of Wilk's Lambda likelihood ratio criterion, the combined fear assessment measures were significantly affected by both group allocation, ($F(15,94) = 5.65$, $p<.001$), sessions, ($F(9,258) = 18.34$, $p<.001$), and by their interaction, ($F(45,128) = 4.78$, $p<.001$). The results indicated a high degree of association existed between the combined fear assessment measures and group membership, $\eta^2 = 0.830$; and sessions, $\eta^2 = 0.700$. That is, 83% of the variance in the linear combination of the time, approaches and latency measures is accounted for by group membership. The results from the MANOVA analysis confirmed the findings of the univariate tests already reported.

A number of analyses of covariance, ANCOVA, were performed to assess the effects of one measure when the other measures are held constant. In the first ANCOVA, the time measure acted as the dependent variable, with the approaches and latency measures acting as covariates. Time spent in the FC compartment varied across group allocation, ($F(5,34) = 5.07$, $p<.002$), across sessions, ($F(3,106) = 20.76$, $p<.001$), and was affected by their interaction, ($F(15,106) = 5.17$, $p<.001$). In the second ANCOVA, the approaches measure acted as the dependent variable, with time and latency measures acting as covariates. Approaches into the FC compartment was significantly affected by both group allocation, ($F(5,34) = 4.59$, $p<.003$), and sessions, ($F(3,106)=19$, $p<.001$), but not by their interaction, ($F(15,106) = 1.54$, $p>.05$). In the final

ANCOVA, the latency measure acted as the dependent variable, with the time and approaches measures acting as covariates. The latency to first enter the FC compartment was significantly affected by group allocation, ($F(5,34) = 3.22$, $p<.02$), sessions, ($F(3,106) = 12.9$, $p<.001$) and by their interaction, ($F(15,106) = 3.56$, $p<.001$. The results of these ANCOVAs indicate that once again all fear assessment measures made unique contributions to the composite dependent variable used in the MANOVA analysis.

In order to explicitly examine the relative sensitivities of the three fear assessment measures in discriminating the propranolol assisted RP treatment groups from the control groups, a stepwise discriminant function analysis was performed. A summary of the results is presented in table 2-34. The analysis yielded three discriminant functions, with a combined Chi Squared of ($X^2(15) = 87.598$, $p<.001$). After removal of the first discriminant function, there still remained a highly significant amount of discriminating power, ($X^2(8) = 33.919$, $p<.001$). After the removal of the second discriminant function, there remained an insignificant amount of discriminating power, ($X^2(3) = 7.674$, $p>.05$), therefore the third discriminant function was dropped from further consideration. The first two discriminant functions accounted for 63.64% and 28.50%, respectively, of the between-groups variability, (see table 2-34(a)).

The canonical discriminant functions evaluated at group means (group centroids) are plotted in figure 2-17. Examining figure 2-17 indicates that the first discriminant function maximally separates PAC, RPP15 and RPP5 groups from RPC subjects, with RPS and RPP10 groups falling between these

TABLE 2-34

(a) Canonical Discriminant Functions - Summary Table

| Function | Eigenvalue | % of Variance | Canonical Correlation | After Function | Wilks Lambda | $X^2$ | D.F. | Significance |
|---|---|---|---|---|---|---|---|---|
| 1 | 0.391 | 63.64 | 0.530 | 0 | 0.583 | 87.598 | 15 | 0.000 |
| 2 | 0.175 | 28.50 | 0.386 | 1 | 0.812 | 33.919 | 8 | 0.000 |
| 3 | 0.048 | 7.86 | 0.215 | 2 | 0.954 | 7.674 | 3 | 0.053 |

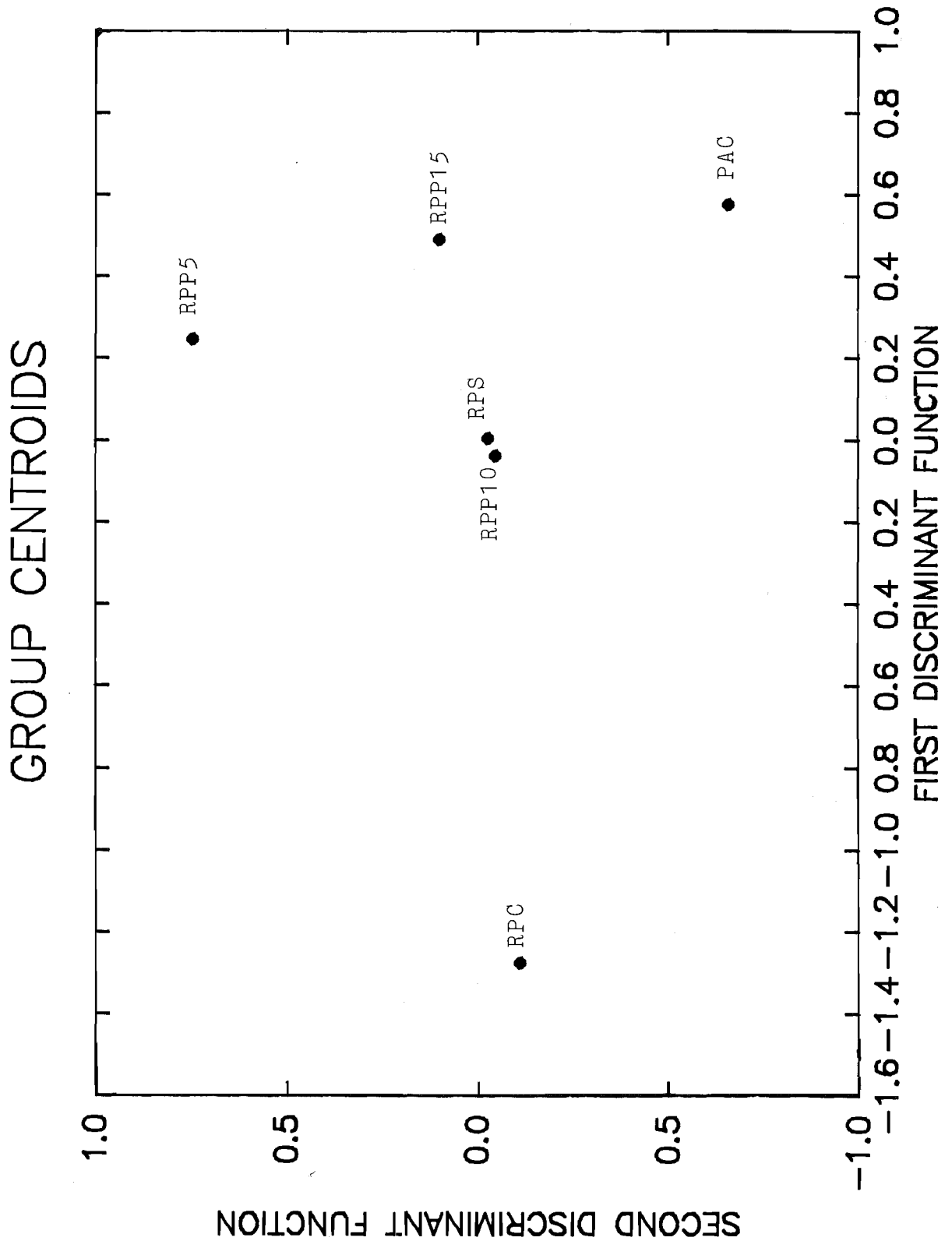
(b) Loading Matrix between predictor variables and discriminant functions.

| Measure | Function 1 | Function 2 |
|---|---|---|
| Time | 0.691 | -0.402 |
| Approaches | 0.584 | 0.746 |
| First entry latency | -0.792 | 0.052 |

(c) Standardised Canonical Discriminant Function Coefficients.

| Measure | Function 1 | Function 2 |
|---|---|---|
| Time | 0.475 | -0.662 |
| Approaches | 0.300 | 0.974 |
| First entry latency | -0.628 | 0.141 |

FIGURE 2-17 Group centroids in the discriminant space formed by the first and second discriminant functions.



groups. The second discriminant function maximally separates RPP5 subjects from the PAC group, with RPC, RPS, RPP10 and RPP15 groups falling between these two groups. Indeed, RPC, RPS, RPP10 and RPP15 groups were indistinguishable from each other on the second discriminant function.

A loading matrix between the measures and discriminant functions, (see table 2-34(b)), indicates that the first discriminant function is correlated most highly with the first entry latency measure, ($r = -0.792$), and was also highly correlated with the time measure, ($r = 0.691$). The second discriminant function is correlated most highly with the approaches measure, ($r = 0.746$), and moderately correlated with the time measure ($r = -0.402$). Relating these findings to the figure of group centroids, it suggests that the maximum spread among the groups on the first discriminant function is primarily based on the first entry latency measure and secondarily based on the time measure. Examining the second discriminant, the groups are distinguished from each other primarily on the basis of the approaches measure, with a moderating influence exhibited by the time measure. Clearly, the approaches measure has failed to distinguish between RPC, RPS, RPP10 and RPP15 groups, although they were distinguished by the first entry latency measure, the first discriminant function.

Examination of the results of the stepping procedure used to enter the measures into the discriminant function analysis indicated that the first entry latency measure had the highest discriminating power of the measures as it entered the analysis first, followed by the approaches measure which in turn was followed by the time measure. All measures

produced significant changes in Rao's V when included in the analysis; for the first entry latency measure (change in Rao's V = 42.75, $p<.001$), for the approaches measure (change in Rao's V = 29.81, $p<.001$) and for the time measure, (change in Rao's V = 27.08, $p<.001$). Therefore, all measures made a significant contribution to the discriminant function analysis.

The above findings suggest that the most sensitive fear assessment measures are the first entry latency and time measures. Although the approaches measures correlated highly with the second discriminant function, it failed to discriminate between the RP treatment groups and RPC subjects.

Two effects of propranolol assisted RP treatment warrant discussion. Firstly, the apparent fearlessness or courage shown by these subjects during session three, and secondly, the transient nature of this behaviour.

The propranolol treated subjects spent more time in the FC compartment during session three in comparison to saline controls. This was most evident with RPP10 subjects, whose individual scores were 97%, 96%, 96%, 91%, 84%, 50% and 22%, of the session time spent in the FC compartment. In comparison, individual scores for the saline controls were 13%, 16%, 21%, 27%, 57%, 62% and 82%. The median time for RPP10 subjects being 91% and for RPS subjects, 27%. The RPP10 rats were not entering the FC compartment and remaining there, they were continuing moving between both compartments, with the mean approaches per minute being 2.12, in comparison with 1.65 for the saline controls. Could the behaviour on the part of propranolol treated subjects be regarded as reflecting courage or fearlessness? Cox, Hallam, O'Connor

and Rachman (1983) have distinguished between courage and fearlessness using Lang's (1970) three-system analysis of fear as follows:

> A person might be quite willing to approach a frightening object or situation, even though he experiences a high degree of subjective fear and unpleasant bodily reactions. Such persistence in the face of subjective and bodily sensations of fear is one definition of courage - to continue despite one's subjective fear ... On the other hand, if a person enters a seemingly dangerous situation but experiences little or no subjective fear or accompanying physiological response, his conduct is correctly described as fearless rather than courageous. (p. 107).

From the Cox et al., viewpoint, then, the propranolol treated subjects were fearless rather than courageous, because following RP treatment it is expected that "subjective" fear and the physiological manifestations of fear are minimal. Yet this effect seemed to be transient, that is, it was not observed during session four. The most parsimonous explanation is that the fearlessness of the propranolol treated subjects represented a state-dependent effect.

State dependent learning, or as it is sometimes called dissocation of learning, (Overton, 1968), refers to the experimental situation where the training and testing is conducted in the placebo or drug conditon, with combinations of these conditions being examined. It is typically reported that learning is superior with the $drug_1$-$drug_1$ combination, in comparison to the placebo-$drug_1$, $drug_1$-placebo, or $drug_1$-$drug_2$ conditions (Overton, 1968, 1974). Another finding also reported, called asymmetrical dissociation, refers to state-dependent learning occuring in the drug-placebo condition,

but not the placebo-drug condition (Barnhart & Abbott, 1967; Berger & Stein, 1969; Overton, 1968). It is proposed that learning decrements are caused by a state change. When a response is learned in a drug or placebo condition, the drug or placebo forms part of the stimulus complex and enters into the control of the response. Thus, moving from the drug state (training) to non-drug state (testing) involves changes in the stimulus complex during training and testing. How might this explain our results? The relatively short-term effect of propranolol in inducing fearlessness, could have been due to the powerful interoceptive drug stimuli during RP treatment providing a context (stimulus complex) for fearless behaviour which persisted for 24 hours (session three) but not for 48 hours (session four). That is, the same drug associated stimulus complex was present during RP treatment and session three, but had changed from a drug-stimulus complex (session three) to a non-drug stimulus complex (session four), and hence, a resultant decrease in fearless behaviour during session four. Clearly, further research using the classic design for evaluation of state-dependent learning (Grosman & Miller, 1961) is required to test the adequacy of this explanation.

## General Discussion

### Major Findings of Experiments One to Four

Experiment one examined the suitability of the escape-from-fear (EFF) active avoidance response as a fear assessment measure within the RP paradigm. The EFF response failed to be sensitive to RP treatment effects. Again, as is common

within the conditioned avoidance literature, the EFF latency raw scores exhibited some heterogeniety of variance, mainly due to a minority of subjects recording maximum EFF latencies on some trials in comparison to the majority of subjects performing with short EFF response latencies. Although the raw scores were transformed to eliminate this heterogeniety of variance, it is possible that the between-subjects variability within treatment groups acted to mask possible RP treatment effects. Clearly, further parametric investigation is necessary to elicit the mechanisms responsible for this within-group variability. One profitable change to the RP procedure of experiment one would have been the presence of nonfearful conspecifics during RP treatment. This was found to increase the efficacy of RP treatment in experiment three.

During experiment one, although the RP treatment session duration was 50 and 55 minutes for RP-100 and RP-200 groups, respectively, the actual non-reinforced CS exposure duration was only 8.33 and 16.67 minutes respectively for each group. Clearly, non-reinforced CS exposure duration may have been insufficient for EFF performance. Rather than examine these changes to the EFF procedure it was decided to modify the EFF procedure to permit conditioned fear assessment using the approach methods developed by Corriveau and Smith (1978).

Experiment two examined the suitability of a modified EFF response procedure as a fear assessment measure within the RP treatment paradigm. While the EFF procedure can be considered as a one-way active avoidance procedure, the modified EFF procedure can be considered as a one-way passive avoidance procedure, amenable to analysis by approach fear

assessment methods. To reiterate a point made earlier, while I have termed the modified EFF procedure a passive avoidance procedure, it is conceptually also an intermittent positive punishment of approaches into the FC compartment procedure. I choose to call the procedure a passive avoidance procedure in order to place emphasis on the response of remaining passive in the safe compartment in order to avoid sampling the shocks presented in the FC compartment.

In experiment two the duration of the RP treatment was varied (1 hour vs 2 hours) as well as its presentation (massed vs distributed). The findings revealed that as the duration of RP increased so did the efficacy of RP treatment in facilitating the extinction of conditioned fear and avoidance, when the fear assessment measures were proportional time spent in the FC compartment and approaches into the FC compartment. This finding is consistent with the research using prolonged RP treatment when conditioned fear is assessed by either active avoidance (Baum, 1968a; Berman & Katzev, 1972; Mineka & Gino, 1979a; and Schiff, et al., 1972) or the CER assessment procedure (Rohrbaugh, et al., 1972; Monti & Smith, 1976; Mineka & Gino, 1979b; Starr & Mineka, 1977) or approach assessment procedures (Bersh & Paynter, 1972; Corriveau & Smith, 1878; Mineka, et al., 1981).

Experiments three and four examined two adjuncts to RP treatment, namely social facilitation and psychopharmacological agents. Social facilitation, the presence of a nonfearful conspecific during RP treatment, increased the efficacy of RP treatment. How might this effect by explained?

The social facilitation effect has been reported in a number of different species across a wide variety of settings

(Guerin & Innes, 1982, 1984; Saunders, 1981). Two explanations of social facilitation seem pertinent to the present discussion. The first explanation suggests that the mere presence of a nonfearful conspecific increases arousal in the experimental subject (Zajonic, 1965, 1980). Zajonic has incorporated the Hull-Spence drive model (Spence, 1956) to account for this increase in arousal. This increase in arousal, could have functioned to move the experimental subject around the FC compartment, thus exposing the subject to the most salient contextual cues associated with the FC compartment. Under the conditions of non-reinforced CS exposure during RP treatment, this increased movement around the FC compartment, as a byproduct of increased arousal, could have facilitated conditioned fear and avoidance extinction.

A second explanation posits that socially mediated fear reduction results from the nonfearful conspecific distracting attention of the experimental subject away from the aversive environmental cues (Moore, Byers & Baron, 1981). This explanation also posits that the more attention the conspecific receives from the experimental subject, the greater the magnitude of fear reduction.

The conspecific's novelty and extent to which it is active and interacting with the experimental subject affects fear reduction. The more novel and more active the conspecific the greater the fear reduction. In the present study the conspecific was novel to the experimental subject and was observed to be engaging in social interaction with the subject as it moved around the FC compartment. Also, there is evidence that novelty of the conspecific increases alertness

and arousal in experimental subjects in comparison to the presence of familiar conspecifics (Guerin & Innes, 1982). Thus, it would seem the results of the present study are comparable with the predictions derived from the distraction hypothesis of social facilitation.

Finally, the above two explanations of social facilitation are probably modulated by the communication between the nonfearful conspecific and experimental subject. As Moore et al., (1981) put it:

> Olfactory cues, auditory signals, and motor behaviour may all carry meaning such that "calm" cues signal safety and "excitatory" cues signal danger. Such a communication mechanism could easily develop via classical conditioning. (p. 487).

The above explanations of the social facilitation effect reported in the present study are not necessarily mutually exclusive, indeed, all these factors may have made a contribution to the obtained effect.

The effects of the benzodiazepine, diazepam, and beta-adrenergic blocking agents, propranolol and atenolol, were complex. One possible explanation to account for these effects is as follows.

Modified two-factor theory proposes that the acquisition and maintenance of avoidance behaviour is mediated by conditioned fear. Two associative links are assumed in the mediational chain:

(1) a CS - conditioned fear (CR) link is established through Pavlovian fear conditioning, and

(2) a CR - avoidance response (Rav) link is established through operant conditioning, with the Rav reinforced via escape/avoidance of the Pavlovian US with accompanying

fear reduction.

The learning of these associative links results from a complex interaction between a number of factors, such as, the contextual cues associated with the conditioning environment (McAllister, et al., 1979; Odling-Smee, 1975, 1978; Testa, 1974); the serial arrangement of CSs along a temporal dimension (Levis, 1981; Stampfl, 1960); species specific reactions to the conditioning process (Bolles, 1970, 1975, 1978; Crawford & Masterton, 1982) and the subject's prevailing expectancies concerning response-reinforcer outcomes during conditioning and subsequent exposure to the CS complex (Seligman & Johnston, 1973; Reiss, 1980).

What are the Concomitants of Conditioned Fear?

Lang (1968, 1971, 1978) has lucidly argued that fear involves three components:physiological, behavioural and subjective. These components may be correlated at the time of measurement (Concordant) or uncorrelated (discordant). They maybe correlated over time (syncronous) or uncorrelated (desynchronous), (Rachman & Hodgson, 1974; Hodgson & Rachman, 1974). The fearlessness shown by the propranolol treated subjects represents concordance between the three components according to a Langian analysis. Thus, during the fear conditioning process there is an interplay between a number of factors with the resultant behavioural outcome representing the interaction between physiological, behavioural and subjective components. In other words, when we look into the behavioural mirror, for example, following fear conditioning and/or RP treatment, the image we see is not merely behavioural, but rather a complex interaction between three

components of fear, which can co-vary or vary independently or change at different rates, which in turn is the result of an interplay between a number of factors during (i) the conditioning procedure and (ii) the subject's subsequent experience of these factors. Also, when we look into the physiological mirror, e.g., via direct measurement of physiological responses, the image we see is not purely physiological, but rather the interaction between the physiological, behavioural and subjective components of fear. One major difference between the Langian three-system analysis of fear and the present proposition, is that Lang does not entertain the existence of motivational-associative mechanisms, while the present proposition does.[1] The above proposition pertains primarily to the data of the present study. This is not to deny the possibility that it can extrapolated to humans to explain fear motivated neurotic behaviours.

While the possibility of conditioned fear acquisition occuring indirectly via vicarious and/or informational mechanisms exists (Rachman, 1977, 1978), the focus on conditioning mechanisms seems most appropriate for the present discussion. Also, in rats, it is difficult to ascertain what the subjective component of a three system analysis of fear represents. Possibly it is best considered as functionally representing some composite of the behavioural and physiological components of fear.

Another difference between the Lang position and the

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[1] For an excellent discussion of associative and non-associative processes in avoidance learning see Anisman (1978).

position offered here is that Lang maintains that the three components of fear are largely independent, which was also noted by Rachman (1978), while the proposition presented here stresses the inter-relationships between the three components of fear.

What is the Functional Utility of Conditioned Fear?

I have proposed the existence of some hypothetical construct or state called conditioned fear. It is further postulated that conditioned fear acts as a source of motivation, because once acquired it affects behaviour in a manner similar to that of known motivators, such as hunger and thirst.

Conditioned fear can be regarded as having two motivational roles. When acquired it energizes behaviour by either permitting escape/avoidance from/of fear-eliciting cues or by suppressing ongoing behavioural activity. A similar view of fear has been proposed by Bolles and Fanselow (1980). When fear is reduced it has a reinforcing property, as evidenced by escape/avoidance learning or the attenuation of response suppression in a CER paradigm. The results of the escape from fear experiment in the present study (experiment one) illustrate the motivational roles of conditioned fear.

Blampied and Kirk (1983) made reference to the motivational role of fear in organizing and co-ordinating defensive behaviour as the basis for explaining the effects of diazepam and oxprenolol on conditioned defensive burying behaviour. They stated:

If it is assumed that burying is defensive, then

> it may reasonably be regarded as motivated by a central motivational state of fear ... if it (burying) is fear motivated, than a centrally acting anxiolytic drug should reduce the central motivational state of fear and consequently reduce fear. Diazepam, a centrally acting anxiolytic, has this effect. In other circumstances, however, fear and anxiety may be disabling, preventing or interfering with the performance of a coping or defensive behaviour (Sluckin, 1979). Again, it is reasonable to suppose that it is primarily the peripheral mainfestations of fear and anxiety which have this disabling effect. A drug which acted to reduce these peripheral symptoms may then facilitate the performance of coping responses. (pp. 698-699).

The present proposition is compatible with the view taken by Blampied and Kirk (1983). Before returning to a discussion of the results from experiments one to four, I shall briefly summarize the proposed underlying mechanisms for the anxiolytic action of benzodiazepines and beta-adrenergic blockers. This seems warranted given the action of these compounds on the biochemical and physiological concommitants of fear, and the relationship of this component of fear to the behavioural and subjective components of fear.

## The Benzodiazepine-Gamma-Aminobutyric Acid (GABA) Connection

Recently, advances in the understanding of the mechanism of action of benzodiazepines have been reported with the discovery by two research groups of specific benzodiazepine receptors located on neuronal membranes in the central nervous system (Mohler & Okada, 1977; Squires & Braestrup, 1977). Subsequent investigation revealed that the benzodiazepine

receptor (BZ) was functionally linked to GABA recognition sites and together they formed a GABA-BZ-chloride ionophore complex (Guidotti, 1981; Paul, Marangos & Skolnick, 1981; Tallman, Paul, Skolnick & Gallager, 1980). The evidence suggests that occupancy of the BZ is not necessary for anxiolytic action (Braestrup & Neilsen, 1980; Gray, 1977), but rather that the CNS actions of benzodiazepines are mediated by the facilitation of GABA transmission (Costa & Guidotti, 1979; Paul, et al., 1981; Tallman, et al., 1980). GABA is the major inhibitory neurotransmitter in the CNS (Snyder, 1984). While a functional linkage appears to exist between BZ and GABA receptors, a structural linkage has yet to be obtained (Lippa, et al., 1980). The overall effect of this mechanism of action for the benzodiazepines is extremely complex. Facilitated GABA transmission results in reduced production of other CNS neurotransmitters, acetylcholine, dopamine and 5-hydroxytryptamine (5-HT), (Collinge, et al., 1982; Iversen, 1978).

This relationship lead Collinge, et al., (1982) to propose that the anxiolytic action of benzodiazepines might be a function of reduced 5-HT production which resulted from increased GABA transmission. It is known that a number of CNS neurotransmitter systems are involved in the regulation of anxiety/fear. The involvement of the nor-adrenergic system has been studied for the last twenty years (Barchas & Freedman, 1963; Corrodi, et al., 1971). The serotonergic (5-HT) system has also been implicated in the psychobiology of anxiety and fear reactions (Sepinwall & Cook, 1980; Stein, Wise & Belluzi, 1975). It is clear that the psychobiology of anxiety and fear represents the

interplay between these neurotransmitter systems although the functions and importance of each system is unknown (Gray, 1981; Sepinwall & Cook, 1981). Although, recently Insel et al., (1984) proposed that the nor-adrenergic and benzodiazepine receptor models of anxiety possibly represented two different clinical phenomena. They stated:

> ... these two proposed pharmacologic models of "anxiety" may correspond to different clinical phenomena: nor-adrenergic activation originating in the pons and corresponding to "alarm", the benzodiazepines system with its dense telencephalic representation corresponding more to "fear" or "conflict".

Further investigation is required to test the validity of this proposition.

The Locus Coeruleus and the Behavioural Inhibition System

Of particular relevance to the present study is the work of Redmond and others in isolating the relationship between anxiety and the locus coeruleus (Redmond, 1977; Redmond & Huang, 1979; Davis, Redmond & Baraban, 1979). The locus coeruleus contains about 50% of the central nor-adrenergic neurons while producing about 70% of nor-adrenaline located centrally. Electircal stimulation of the locus coeruleus in monkeys (Redmond & Huang, 1979) and humans (Nashold, Wilson & Slaughter, 1974) produces reactions identical to those of "fearlike" behaviours. This reaction has also been reported with drugs known to increase locus coeruleus activity, piperoxanhydrochloride and yohimbine, in monkeys (Redmond & Huang, 1979) and humans (Soffer, 1954; Holmberg & Gershon, 1961). The opposite effect, an anxiolytic action,

has been reported with drugs that decrease locus coeruleus activity, clonidine hydrochloride and diazepam, in monkeys (Redmond & Huang, 1979) and humans (Charney, Heninger & Redmond, 1983). Propranolol has also been reported to partially block the effects of electrical stimulation of the locus coeruleus (Redmond & Huang, 1979). Redmond (1977) proposed a possible neurophysiological model of anxiety centred on the action of the locus coeruleus. In summarizing the interactions between the CNS and ANS systems, he stated:

> Activation of the locus ceruleus-cortical or limbic efferent projection areas would lead to coping strategies, or defenses, to remove the threat ... The locus ceruleus may be involved in neuroendocrine changes associated with stress and anxiety ... Modulation of arousal state may be an interactive function with other systems in the pontine reticular formation and elsewhere ... The caudal outflow may be responsible for cardiovascular and sympathetic changes associated both with anxiety and stimulation of the locus ceruleus. These effects are interactive with the parasympathetic system to produce the particular visceral manifestations of anxiety. Enteroception of these peripheral changes feeds back via the cortex and, perhaps, to the locus ceruleus directly, in a feedback loop, the blockade of which has been interpreted as the mode of anti-anxiety action of the B-adrenergic antagonist propranolol. (p. 298).

Redmond's model provides the possible mechanism for the anxiolytic actions of beta-adrenergic blocking agents. A similar, but more far reaching proposition for explaining the psychobiology of anxiety/fear has been presented by Gray (1977, 1982). Gray proposed a behavioural inhibition system as a model for anxiety/fear and the action of anxiolytic

drugs, which implicates the septohippocampal system and its dorsal afferents as the neural substrate of anxiety/fear. Gray (1977) reported that anxiety and fear responses in animals produced a specific hippocampal theta rhythm, 7.7Hz, which could be modified by anxiolytic drugs, but only when the connections between the locus coeruleus and the septum are intact. This provided evidence for a functional linkage between the nor-adrenergic and limbic systems. Limbic system structures, the hippocampus and amygdala, have high densities of benzodiazepine receptors (Young & Kuher, 1980), indicating the limbic system as a possible site of action for the benzodiazepines.

It is clear from the foregoing discussion that the mechanisms of action of benzodiazepines and beta-adrenergic blockers are extremely complex. The psychological and somatic symptoms of anxiety and fear are probably mediated by different neurotransmitter systems, with their inter-relationships determining the action of anxiolytic drug compounds. Whether beta-adrenergic blocking agents produce their anxiolytic effect by peripheral beta-adrenergic receptor blockade or by a central effect possibly mediated through changes in locus coeruleus activity, is still unresolved, although the experimental evidence supports primarily a peripheral beta-adrenergic receptor blocking explanation, with secondary central effects (Searle, 1983).

The present study employed a number of behavioural measures to assess the effects of various independent variable manipulations on conditioned fear and avoidance behaviour. The three fear assessment measures, time, approaches and latency, correlated reasonably well, but not

perfectly. This suggests that the fear assessment measures were sensitive to a number of non-redundant components of conditioned fear and psychophysiological processes occurring during avoidance acquisition and extinction. How might these fear assessment measures by interpreted?

The First Entry Latency Measure

The latency measure represented the time the subject took to initiate motor responses directed towards and into the FC compartment. Any process which suppresses the initial instantiation of motor activity and movement directly affects the latency measure, by producing an increase in the first entry latency measure. Pavlovian fear conditioning during the second session had this effect on the latency measure. This is illustrated with reference to the RPC and CSC subjects of experiment three. During session three, 4 of the RPC subjects failed to enter the FC compartment, indicating substantial inhibition of motor activity towards and into the FC compartment. Similarily, during day 3, when RP subjects received RP treatments, the CSC subjects were placed into the apparatus and permitted free access to both compartments, and 5 of the 7 subjects failed to enter the FC compartment. In comparison, non-avoidant, fear conditioning control subjects, PAC, all entered the FC compartment during session three, with the longest first entry latency being 7 seconds. A parsimonous explanation for these changes in first entry latency is that conditioned fear inhibited the initiation of active motor response behaviours. This motivational effect of conditioned fear could have been reinforced by the expectancy that approach and entry into

the FC compartment would produce aversive consequences, US delivery, since both RPC and CSC received no disconfirmation of this response-outcome expectancy through non-reinforced CS exposure in the FC compartment. The CSC subjects failed to voluntarily expose themselves to the CS and contextual cues of the FC compartment during day 3 of experiment three when they had the opportunity to do so. Also, movement cues associated with activity around the safe compartment, through response and stimulus generalization, could have been associated with the response produced stimuli of the CS-complex present during the US delivery phase of fear conditioning, thus, further reinforcing the inhibition of motor activity. Such movement would interact with conditioned fear inhibiting further movement directed towards entry into the FC compartment. If this is the case, then each segment of motor activity directed towards the opening between the compartments and subsequent withdrawal from the opening could be conceptualized as a Pavlovian extinction trial. It is also comparable to the safety-testing behaviours observed by Corriveau & Smith (1978). If such behaviour represents a Pavlovian extinction trial, then repetitions of approach and withdrawal responses would lead to conditioned fear reduction associated with this part of the CS-complex following continued non-reinforcement. Evenutally, the subject would approach and enter the FC compartment. Any process or procedure that decreased fear associated with the CS and contextual cues of the FC compartment would produce disinhibition of active motor behaviour and shorten the first entry latency. The response prevention treatments of the present study had this effect.

Blampied and Kirk (1983) proposed that conditioned fear can be disabling and intefer with coping behaviour. The disabling effects of fear are primarily a function of the peripheral manifestations of fear and anxiety. Given this, it might be expected that beta-adrenergic blockers, whose anxiolytic action is believed to be primarily by peripheral beta-blockade, would alleviate the disruption of ongoing motor activity. This was partially confirmed by the results of experiments three and four. The results of diazepam-assisted RP treatment, where diazepam is a centrally acting anxiolytic, consistently paralleled those of saline assisted RP treatment subjects. In comparison, the beta-blocker assisted RP treatment subjects typically first entered the FC compartment with shorter latencies than controls, although not significantly so, with the exception of 10.0 mg/kg propranolol subjects. While the first entry latencies were relatively short for 10.0 mg/kg propranolol subjects during session three, (mean = 10 seconds) two subjects failed to enter the FC compartment during session four, resulting in a substantial increase in the group mean latency score, which was 734 seconds. Given that the extreme latency scores of two subjects inflated the mean value and obscured the central tendency exhibited by the majority of scores, medians were calculated for comparison between sessions three and four of experiment three. For session three the median first entry latency was 4 seconds and for session four it was 30 seconds. While an increase in first entry latency is still evident with a median analysis, the increase is not so marked. Yet the increase may represent a centrally motivated process producing freezing and inhibition of motor activity during

session four which was not alleviated by peripheral blockade of the somatic concomitants of fear and anxiety by beta-adrenergic blockers. Also, the increase in first entry latency during session four can be explained as a drug state-dependent effect, with the effect being strongest for the subjects who failed to enter the FC compartment.

While the results of experiment three support a peripheral beta-blockade mode of action for the beta-blocker effects on first entry latency, some central influences cannot be ruled out. Firstly, if the anxiolytic effects were primarily peripheral there should have been little difference between propranolol and atenolol treated subjects, but there was. Atenolol treated subjects had shorter first entry latencies in comparison to propranolol treated subjects. Possible explanations for the propranolol effect have already been discussed, but the existence of a difference between these beta-blockers in their effects suggests a centrally mediated influence on one or both of these drugs, but to different degrees. Secondly, central effects of these drugs have been confirmed in recent work performed in our laboratory by Searle (1983). Searle reported decreased locus ceruleus activity during presentation of an aversive white noise 15 minutes following a 10 mg/kg propranolol injection (i.p.), with a lesser effect reported for a 10 mg/kg atenolol injection (i.p.). Given that both propranolol and atenolol had the same effects on Searle's behavioural measures of fear and anxiety, his findings suggested that beta-blocker anxiolytic activity was primarily due to peripheral beta-blockade with a secondary central affect possibly mediated via changes in locus coeruleus activity, with this secondary

affect being more pronounced for propranolol in comparison to atenolol. The results reported by Searle (1983) are consistent with the proposition of the present study that the primary site of anxiolytic action of the beta-blockers is peripheral beta-blockade with secondary central effects either by direct blockade of beta-adrenergic receptors, by direct changes in locus coeruleus activity, indirect changes mediated by locus coeruleus cortical projections, or a combination of these mechanisms.

Approaches into the FC Compartment Measure

The approaches measure represented the number of times each subject entered the FC compartment during each session. It can be regarded as an expression of normal, nonfearful exploratory behaviour on the part of the subjects. As such, it should be at a maximum during session one decreasing during that session to some asymptotic level. This is what occurred with the non-avoidant, fear conditioning controls, PAC subjects. The approaches measure is only meaningful for behaviourally active subjects, and then only after the first entry into the FC compartment has been performed.

The processes and mechanisms discussed in regard to the first entry latency measure are also applicable to the approaches measure. In addition, in the absence of interpolated RP treatment, approaches into the FC compartment serve to bring about non-reinforced CS exposure and exposure to the contextual cues associated with the FC compartment. This experience facilitates conditioned fear reduction. Also, such behaviour would facilitate disconfirmation of the response-outcome expectancy learned during fear conditioning.

Similarly, following RP treatment, approaches into the FC compartment should further buttress and reinforce the processes in operation during RP treatment.

While the approaches measure was expected to be a sensitive fear assessment measure, it was not as sensitive as the other measures. This was evident from the individual analyses of each measure and also from the discriminant function analyses. In experiment two, both measures, time and approaches, correlated highly, 0.74 and 0.89, respectively, with the first discriminant function of the discriminant function analysis. The discriminating ability of the approaches measure in experiment two may have resulted from the contribution of the first entry latency, which was not measured in experiment two but examined in later experiments. In experiments three and four, the latency measure correlated, 0.663 and -0.792, respectively, with the first discriminant function, while the approaches measure in these experiments correlated 0.284 and 0.584, respectively, with the first discriminant function. This result suggests that when the effect of approaches into the FC compartment is differentiated into two components, the first entry latency and approaches into the FC compartment, the first entry latency measure makes a greater contribution to the discrimination between the treatment groups. This conclusion is confirmed by previous research which also reported the superiority of the first approach latency over total number of approaches (Corriveau & Smith, 1978; Corriveau, et al., 1978; Bersh & Paynter, 1972).

Time Measure

The time measure represents the proportion of total session time spent in the FC compartment. The time measure is seen as a function of both the first entry latency and approaches measures. It is negatively correlated with the first entry latency. Thus, the longer the first entry latency the shorter the remaining session time available for entry and exploration of the FC compartment. The time measure is also the summation of the following two behavioural effects: firstly, subjects might enter the FC compartment, remain there for some time, then return to the safe compartment or fail to move back to the safe compartment. Secondly, subjects might move freely between both compartments continuously during the session. Therefore a proportional time score of 0.50, 50% of session time spent in the FC compartment, could reflect a subject moving into the FC compartment, remaining there for 15 minutes, then returning to the safe compartment for the remainder of the session. Alternatively, a score of 0.50 could reflect the behaviour of a subject continuously moving between compartments during a session with the net result indicating that the subject spent half the session time in the FC compartment. The major difference in performance between these subjects is the average time spent in the FC compartment per entry. For the first subject the average was high (15 minutes) for the second subject it was considerably less (say, less than a minute). Might these two different behavioural outcomes represent the effects of different yet inter-related processes?

A subject entering and remaining in the FC compartment

for some time maybe indicative of the situation where the centrally motivated manifestations of fear and anxiety have been reduced or extinguished resulting in the subject being nonfearful of the CS and contextual cues associated with the FC compartment. This could result following RP treatment or administration of centrally acting anxiolytic drugs, such as diazepam. Alternatively, a subject making many enteries into the FC compartment, but only remaining for a short time per entry, might be indicative of the situation whereby the peripheral manifestations of fear and anxiety have been reduced or eliminated, resulting in the initiation of active motor and exploratory responses, but the central manifestations of fear and anxiety are re-activated by entry and exposure to the CS and contextual cues associated with the FC compartment, resulting in withdrawal back into the safe compartment. This state might result following the administration of beta-adrenergic blockers. This explanation for interpretation of the time fear assessment measure receives support from the present study.

In the following discussion reference is made to proportional time scores collapsed across extinction sessions, three and four. In experiment three the saline controls spent 41% of the session time in the FC compartment. They also made 1.70 approaches per minute into the FC compartment. In comparison, diazepam subjects spent an equivalent amount of time in the FC compartment, 43%, but made fewer approaches, 1.30 per minute. This suggests that diazepam subjects spent more time per entry in the FC compartment in comparison to saline controls. This possibly represented a decrease in the central manifestations of fear and anxiety due to

the anxiolytic action of diazepam. In comparison to saline and diazepam groups, atenolol subjects spent less time in the FC compartment, 31%, but made significantly more approaches, 2.19. Clearly, atenolol subjects spent significantly less time per entry in the FC compartment in comparison to both saline and diazepam groups. This possibly represented the blocking of the feedback mechanisms of the peripheral manifestations of fear and anxiety by atenolol, with little effect on the central manifestations. Indicating, again, that the primary site of anxiolytic action for atenolol is peripheral beta-blockade. The subjects administered propranolol (10 mg/kg) spent 54.6% of the session time in the FC compartment, making 1.8 approaches. Propranolol subjects spent more time in the FC compartment in comparison to saline and atenolol groups, making fewer approaches than atenolol, but more than saline subjects. Possibly the behaviour exhibited by propranolol subjects represented an interaction of propranolol's influence on both the central and peripheral manifestations of fear and anxiety.

The above explanation might also account for the differences reported between the dose-response functions of experiment four. At low doses the anxiolytic action of propranolol could be primarily peripheral beta-blockade, while at higher doses the central effects of propranolol became evident with higher concentrations passing through the blood-brain barrier. As the dose level of propranolol increased, the time spent in the FC compartment initially increased then slightly decreased, 38.9% (5 mg/kg), 54.6% (10 mg/kg), and 43.8% (15 mg/kg). On the other hand, approaches per minute decreased consistantly with increasing

propranolol dose level, 2.92, 1.80 and 1.78 respectively. At 5 mg/kg propranolol subjects made the most approaches and spent less time per entry in comparison to the 10 mg/kg and 15 mg/kg dose groups, possibly indicating a peripherally mediated effect of propranolol. Wheras at the 15 mg/kg dose, subjects spent more time per entry in the FC compartment in comparison to 5 mg/kg and 10 mg/kg dose groups, possibly reflecting the interaction of the peripheral and central actions of propranolol at that dose level.

The results of the univariate, multivariate and discriminant function analyses revealed that the time measure was sensitive in discriminating between the treatment groups, and thus, a sensitive fear assessment measure. The discriminant function analyses revealed that the time measure correlated highly with the first discriminant function in all passive avoidance experiments, 0.743 (experiment two), 0.809 (experiment three) and 0.691 (experiment four). The present findings therefore confirmed previous research which reported that time spent in contact with the stimuli associated with aversive conditioning acted as a sensitive fear assessment measure (Mineka, et al., 1981; Miller, et al., 1982).

In summary, the foregoing discussion has focused on the view that the acquisition, maintenance and extinction of avoidance behaviour is a function of associative and nonassociative processes (see Anisman, 1978), which are modulated by such factors as the contextual cues associated with the conditioning environment, species specific reactions to the environment and the subject's response-outcome expectancies. In turn, these factors impinge upon three components of fear, behavioural, physiological and subjective.

Together, their complex interplay and interaction results in the observed central and peripheral manifestations of fear and anxiety. It is within this context that the present study's findings were discussed.

## Theoretical Accounts of the RP Treatment Results

The findings of the present study support modified two-process learning theory. If complete extinction of conditioned fear takes place during RP treatment, then residual fear following RP treatment should be absent. This was reported for experiment three. Increasing the duration of RP treatment should increase its efficacy, according to modified two-process theory. This finding was reported for experiment two.

The findings of the present study also support Seligman and Johnston's (1973) cognitive theory of RP treatment. However, as previously discussed, this theory's major weakness is its lack of testable boundary conditions. Whenever an RP treatment effect is obtained it is explicable by cognitive theory. Conversely, whenever failure to obtain an RP treatment effect is reported, this is also explicable by cognitive theory. The difficulty for cognitive theory is to independently assess changes in cognitive expectancies. In other words, it is extremely difficult to assess whether changes in cognitive expectancies are the product or cause of conditioned fear.

The present study failed to support the competing response theory of RP treatment. Briefly, this theory posits that RP treatment eliminates avoidance responding leaving the underlying conditioned fear unaltered. Thus, conditioned

fear motivates new competing responses such as immobility. Since residual fear was low or eliminated in the present study this is incompatible with a competing response analysis. Also, using approach assessment measures, if a competing response such as freezing had developed during RP treatment, then RP treatment group subjects should exhibit a strong tendency to freeze and remain immobile during the fear assessment phases of the experiments, i.e., the sessions following RP treatment. This did not happen as RP treatment groups spent more time in the FC compartment, performed more approaches into the FC compartment, and had a shorter latency to first enter the FC compartment in comparison to RP treatment control subjects. Indeed, it was the subjects not receiving RP treatment that remained immobile in the safe compartment during the conditioned fear assessment sessions.

The findings of experiments three and four are particularly relevant to Baum's relaxational analysis of RP treatment. Briefly, relaxation theory posits that during RP treatment, neither Pavlovian extinction of conditioned fear occurs nor does the subject acquire a competing response. Rather, RP treatment eliminates fear-motivated behaviour because the subject learns to relax. Previous research has demonstrated that relaxational processes can be induced by techniques used as adjuncts to RP treatment (Lederhendler & Baum, 1970; Baum, 1969c). One such technique is social facilitation. The findings of the present study, experiment three, where 1 hour RP treatment in the presence of a nonfearful conspecific was more effective in reducing conditioned fear and avoidance than 1 hour alone during RP treatment supports the

relaxational analysis of RP treatment. However, the findings of the psychopharmacological interventions were less consistent with a relaxational analysis. According to a relaxation analysis, the use of tranquilizing, anxiolytic agents as adjuncts to RP treatment should increase the efficacy of RP treatment. A number of studies, reviewed previously, and the present study, have failed consistently to report reliable, significant effects of anxiolytic agents. While the present study produced effects in the direction of increased efficacy of RP treatment by diazepam, propranolol and atenolol, these effects were transient in nature. That is, they were present during the first fear assessment session but had disappeared by the second fear assessment session. The results of the present study indicated, that while relaxational processes during RP treatment are desirable for conditioned fear and avoidance reduction, they alone were insufficient to produce reliable and enduring facilitated RP treatment effects. The present study examined the effects of two beta-adrenergic blockers, propranolol and atenolol. Propranolol, a non-selective beta-blocker, passes easily across the blood-brain barrier. Atenolol, a cardioselective beta-blocker, is believed to pass poorly from the blood stream into the brain.

If Atenolol fails to pass through the blood-brain barrier in any significant quantity, but is effective in producing conditioned fear and avoidance extinction, then peripheral beta-adrenergic blockade must be responsible for this anxiolytic action. However, if an anxiolytic effect is produced by propranolol, but not atenolol, then the anxiolytic effect can most reasonably be referred to the

action of propranolol centrally. While the majority of beta-adrenergic blockers can pass through the blood-barrier and exert their influence centrally, research evidence suggests that their anxiolytic action primarily focuses on blockade of the peripheral feedback mechanisms from the somatic components of conditioned fear (Keilholz, 1977, 1978).

In comparing the effects of propranolol and atenolol assisted RP treatments, the only significant comparison was for the proportional time spent in the FC compartment during session three, where propranolol treated subjects spent significantly more time in the FC compartment. The atenolol treated subjects performed more approaches into the FC compartment and had a shorter latency to first enter the FC compartment in comparison to propranolol treated subjects. Although these results need to be interpreted with some caution, it would seem that they have supported the proposition that beta-adrenergic blockers exert their anxiolytic action primarily through their peripheral action, rather than their central influences, which confirms previous findings from our laboratory (Blampied & Kirk, 1983; Hughes, 1981). But further research, with a broader range of dose-levels across more beta-blocking agents, is required to achieve a clearer picture of the anxiolytic actions of beta-adrenergic blockers.

The possible weakness of the current study was that the assessment of the effects of nonfearful conspecifics and beta-adrenergic blockers on RP treatment and conditioned fear reduction was confounded. That is, the drugs were injected prior to RP treatment with the presence of a nonfearful conspecific, the two variables were not assessed

independently of each other. It is therefore possible that drug assisted RP treatment failed to be superior to RP treatment controls because of the effect of the nonfearful conspecific during RP treatment. Given that there is a maximum limit on the amount of relaxational behaviours performed during RP treatment, it is possible that the presence of a nonfearful conspecific produced a ceiling effect with respect to relaxational behaviours. This could have masked the anxiolytic actions of beta-adrenergic blocker assisted RP treatment. Clearly, the present study should be replicated with the use of beta-blocker assisted RP treatment with and without the presence of a conspecific.